A Comparative study of Epidural 0.5% Isobaric Levobupivacaine and Epidural 0.5% Isobaric Levobupivacaine with Dexmedetomidine for patients undergoing elective infraumbilical and lower limb surgeries

Dissertation submitted

in partial fulfillment of the requirements

for the award of the Degree

M.D. (Anaesthesiology)

Branch X

GOVT. KILPAUK MEDICAL COLLEGE

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY CHENNAI, TAMILNADU

APRIL 2015

CERTIFICATE

This is to certify that this dissertation entitled "**A Comparative study of Epidural 0.5% Isobaric Levobupivacaine and Epidural 0.5% Isobaric Levobupivacaine with Dexmedetomidine for patients undergoing elective infraumbilical and lower limb surgeries** submitted by Dr.MANGAL SWATHI.V in partial fulfillment for the award of the Degree Doctor of Medicine in Anaesthesiology by The Tamil Nadu Dr. M.G.R. Medical University, Chennai is a bonafide work done by her at GOVERNMENT KILPAUK MEDICAL COLLEGE, during the academic year 2012-2015.

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DECLARATION

I, Dr. MANGAL SWATHI.V, solemnly declare that this dissertation, entitled "**A Comparative study of Epidural 0.5% Isobaric Levobupivacaine and Epidural 0.5% Isobaric Levobupivacaine with Dexmedetomidine for patients undergoing elective infraumbilical and lower limb surgeries"** has been prepared by me, under the expert guidance and supervision of **PROF.Dr.T.MURUGAN,M.D.,D.A.,** Professor & HOD, Department of Anaesthesiology, Government Kilpauk Medical College ,Chennai and submitted in partial fulfillment of the regulations for the award of the Degree M.D.(Anaesthesiology) by The Tamil Nadu Dr. M.G.R. Medical University and the examination to be held in April 2015.

This study was conducted at Government Kilpauk Medical College Hospital and Government Royapettah Hospital, Chennai. I have not submitted this dissertation previously to any university for the award of any Degree or Diploma.

Place: Chennai **(Dr.MANGAL SWATHI.V)**

Date:

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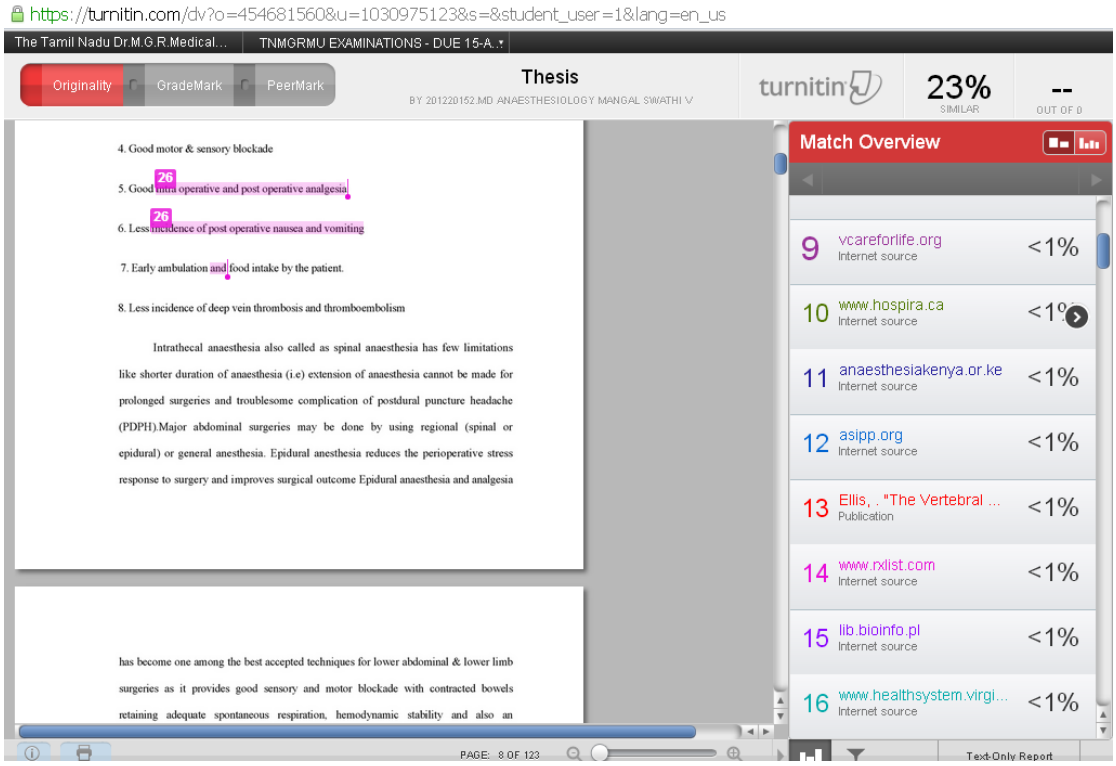
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4. Good motor & sensory blockade
5. Good intra operative and post operative analgesia
6. Less incidence of post operative nausea and vomiting
7. Early ambulation and food intake by the patient.
8. Less incidence of deep vein thrombosis and thromboembolism
Intrathecal anaesthesia also called as spinal anaesthesia has few limitations like shorter duration of anaesthesia (i.e) extension of anaesthesia cannot be made for prolonged surgeries and troublesome complication of postdural puncture headache (PDPH).Major abdominal surgeries may be done by using regional (spinal or epidural) or general anesthesia. Epidural anesthesia reduces the perioperative stress response to surgery and improves surgical outcome Epidural anaesthesia and analgesia
has become one among the best accepted techniques for lower abdominal & lower limb surgeries as it provides good sensory and motor blockade with contracted bowels retaining adequate spontaneous respiration, hemodynamic stability and also an
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ABSTRACT

BACKGROUND

The quest for searching newer and safer anaesthetic agents has always been one of the primary needs in anaesthesiology practice. Regional anaesthesia techniques have seen numerous modifications over the last two decades with the advent of many newer and safer local anaesthetics

Keeping these factors in mind, S (−)-enantiomer of bupivacaine, levobupivacaine has been developed. The advantages of levobupivacaine over bupivacaine are decreased cardiovascular toxicity and there is also a relatively decreased motor nerve fiber penetration and block, thereby a decreased post operative motor blockade and thus early ambulation of the patients can be achieved.

The present study compared the effects of addition of epidural dexmedetomidine 50 micrograms to epidural 0.5% levobupivacaine for infraumbilical and lower limb surgeries.

METHODS

Sixty patients of either sex belonging to ASA I & II in the age group of 25-45 years scheduled for infraumbilical and lower limb surgeries were randomly divided into 2 groups (30 each)to receive 0.5% isobaric levobupivacaine 20 ml epidurally with 0.5 ml distilled water(Group A) and 0.5% isobaric levobupivacaine 20 ml plus 0.5 ml dexmedetomidine containing 50 micrograms(Group B).

This study evaluated the following parameters like time of onset of sensory blockade at T10 level, maximum sensory blockade achieved and time taken to achieve the same, onset time of motor blockade, degree of motor blockade, time taken to achieve maximal motor blockade, hemodynamic changes in pulse rate, blood pressure and oxygen saturation, side effects and complications, intraoperative sedation scores, duration of analgesia, sensory & motor blockade, and any postoperative adverse reactions.

RESULTS

The data obtained from the above parameters were statistically analysed using SSPS version 16 software. Student t test was used for parametric data and Chi-square test for non parametric data.$P<0.05$ was considered as statistically significant.

Maximal sensory level was achieved with addition of dexmedetomidine ranging from T4 to T6.Also the onset time of motor blockade was shortened with group A showing 19.33 minutes and group B showing only 14.5 minutes.The maximal motor blockade achieved was also intense(Bromage 3) with the addition of dexmedetomidine. Duration of analgesia, sensory and motor blockade were prolonged when levobupivacaine is combined with dexmedetomidine epidurally. Changes in hemodynamic parameters (blood pressure & heart rate) were very

minimal in the dexmedetomidine group. Adverse effects experienced in general were statistically insignificant in both the groups. Mean sedation score in group B (Dexmedetomidine group) was predominantly found to be 2 as per Ramsay sedation score. None of the patients in group B had deep sedation or profound respiratory depression.

KEYWORDS: Epidural anaesthesia, Levobupivacaine, Dexmedetomidine, infraumbilical surgeries, lower limb surgeries.

INTRODUCTION

Regional anaesthesia came in vogue from the time of Sir August Bier in 1898. Intrathecal anaesthesia and epidural anaesthesia are the most popular regional anaesthetic techniques used for lower abdominal & lower limb surgeries. Regional anaesthesia forms an excellent alternative due to its various advantages over general anaesthesia including[1]

1. Awake patient

2. Polypharmacy avoided

3. No airway manipulation

4. Good motor & sensory blockade

5. Good intra operative and post operative analgesia

6. Less incidence of post operative nausea and vomiting

7. Early ambulation and food intake by the patient.

8. Less incidence of deep vein thrombosis and thromboembolism

Intrathecal anaesthesia also called as spinal anaesthesia has few limitations like shorter duration of anaesthesia (i.e) anaesthesia cannot be maintained for prolonged surgeries and troublesome complication of postdural puncture headache (PDPH).Major abdominal surgeries may be done by using regional (spinal or epidural) or general anaesthesia. Epidural anaesthesia reduces the perioperative stress

response to surgery and improves surgical outcome.Epidural anaesthesia and analgesia has become one among the best accepted techniques for lower abdominal & lower limb surgeries as it provides good sensory and motor blockade with contracted bowels retaining adequate spontaneous respiration, hemodynamic stability and also an indwelling epidural catheter facilitates further administration of analgesic doses for the postoperative period[2].

Advantages of epidural over spinal anaesthesia being that it [3]

1. Reduces the incidence of hemodynamic changes due to sympathetic blockade as it produces segmental anaesthesia unlike subarachnoid block.

2. Provides effective surgical anaesthesia for prolonged procedures.

3. Incidence of postdural puncture headache is not encountered as dura is not pierced.

4. Provides prolonged postoperative analgesia.

Lignocaine, a tertiary amide local anaesthetic was synthesised by Nils Lofgren in 1943[3].Use of lignocaine for extradural blockade was found to have a greater speed of onset and greater degree of both sensory and motor blockade [4].

But it is also observed that bupivacaine is four times more potent than lignocaine and duration of anaesthesia is 2 or 3 times longer than Lignocaine. It has excellent prolonged duration of sensory blockade, but muscle relaxation was not profound[5,6]

Bupivacaine was synthesised by Ekanstam in 1956 and introduced into clinical practice in 1963 by Telivuo. Bupivacaine, the widely used local anaesthetic in regional anaesthesia is available in a commercial preparation as a racemic mixture (50:50) of its two enantiomers levobupivacaine, S (-) isomer and dextrobupivacaine, R (+) isomer. Among the amide local anaesthetics used in regional anaesthetic techniques bupivacaine has emerged as the most commonly used drug for central neuraxial blockade. Since then bupivacaine is extensively used and has become very popular for epidural anaesthesia as well as analgesia because of its long duration of action. Despite its undoubted efficacy, bupivacaine is associated with cardiotoxicity and neurotoxicity[7,8].

Severe neurotoxic and cardiovascular adverse reactions reported in the literature after inadvertent intravascular injection or intravenous regional anaesthesia have been linked to the R(+) isomer of bupivacaine. The levorotatory isomers were shown to have a safer pharmacological profile with less cardiac and neurotoxic adverse effects.[7,8]Apart from cardiotoxicity, it also carries undesirable effects like prolonged post operative motor blockade. Hence there was a need for introduction of drugs with all the features of bupivacaine but having less toxicity profile.[9]

The quest for searching newer and safer anaesthetic agents has always been one of the primary needs in anaesthesiology practice. Regional anaesthesia techniques have seen numerous modifications over the last two decades with the advent of many newer and safer local anaesthetics[10,11].

Keeping these factors in mind, S (−)-enantiomer of bupivacaine, levobupivacaine has been developed. The advantages of levobupivacaine over

bupivacaine are decreased cardiovascular toxicity and there is also a relatively decreased motor nerve fiber penetration and block, thereby a decreased post operative motor blockade and thus early ambulation of the patients can be achieved[9].

It demonstrated less affinity and strength of depressant effects onto myocardial and central nervous vital centres in pharmacodynamic studies and a superior pharmacokinetic profile. Reports of toxicity with levobupivacaine are scarce and occasional toxic symptoms are usually reversible with minimal treatment with no fatal outcome. In anaesthesia and analgesia practice, levobupivacaine and bupivacaine produce comparable surgical sensory block but levobupivacaine has the advantage of producing less motor blockade. [9]

The low cardiovascular and neurological toxicity of levobupivacaine has led to its varied utility in regional anaesthetic techniques including subarachnoid block, epidural anaesthesia and analgesia, brachial plexus blocks as well as local infiltration. It is also being used for intraoperative anaesthesia, labour analgesia, and postoperative pain management. Both levobupivacaine and ropivacaine are being favoured in labour analgesia because of comparable long lasting analgesia, less motor block and less toxicity compared to bupivacaine.[12]

Efforts to find a better adjuvant in regional anaesthesia are underway since long. The addition of adjunctive agents (opioids & alpha 2 agonists) to local anaesthetics via epidural and intrathecal routes may provide a dose sparing effect and increase the duration and quality of analgesia. [9]

Opioids like fentanyl have been used traditionally as an adjunct for epidural administration in combination with lower dose of local anaesthetic to achieve the desired

anaesthetic effect. The addition of opioid does provide a dose sparing effect of local anaesthetic and superior analgesia but there is always a possibility of an increased incidence of pruritis, urinary retention, nausea, vomiting and respiratory depression. [9]

The pharmacologic properties of alpha 2 agonist have been extensively studied and have been employed clinically to achieve desired effects in regional anaesthesia. Clonidine is an alpha-2 adrenergic agonist which, when administered by epidural route, has analgesic properties and potentiates the effect of local anaesthetics. Clonidine is 200-folds more selective for alpha-2 as compared to alpha-1.

Dexmedetomidine is a new addition to the class of alpha 2 agonists which has got numerous beneficial effects when used through epidural route. It acts on both pre and post synaptic sympathetic nerve terminals and central nervous system, thereby decreasing the sympathetic outflow and nor epinephrine release causing sedative, anti-anxiety, analgesic, sympatholytic and hemodynamic effects.[13]

Dexmedetomidine, made up of medetomidine's dextrogyrous enantiomer,is currently considered a super selective alpha-2 adrenergic agonist prototype and is 1600-folds more selective for alpha-2 receptor. Dexmedetomidine is a highly selective alpha 2 adrenergic agonist with greater receptor affinity than clonidine.[9] In addition it also has hemodynamic stabilising effects and reduction of anaesthetic drug requirements. Epidural dexmedetomidine does cause a manageable hypotension & bradycardia but the striking feature of this drug is its lack of opioid related side effects like respiratory depression, pruritis, nausea and vomiting. [14]

Hence the present study is to evaluate the effects of addition of dexmedetomidine to epidural 0.5% levobupivacaine in infraumbilical and lower limb surgeries.

AIM OF THE STUDY

The aim of the present study is to evaluate the effect of addition of Dexmedetomidine to epidural 0.5% Levobupivacaine solution on the

1. Time of onset of sensory blockade at T10 level
2. Maximum sensory blockade and time taken to achieve that level
3. Onset of motor blockade
4. Degree of motor blockade achieved
5. Haemodynamic changes in the pulse rate & blood pressure
6. Side effects and complications
7. Intraoperative sedation scores
8. Duration of analgesia
9. Duration of sensory blockade
10. Duration of motor blockade
11. Any postoperative untoward reactions

ANATOMY OF THE VERTEBRAL COLUMN [15]

Knowledge of the anatomy of vertebral column is of particular importance to anaesthesiologists. There are

7 Cervical vertebrae

12 Thoracic vertebrae

5 Lumbar vertebrae

5 Sacral vertebrae (Fused)

4 Coccygeal vertebrae (Fused)

The cervical and lumbar curvatures are convex anteriorly while, thoracic and sacral curvatures are convex posteriorly.

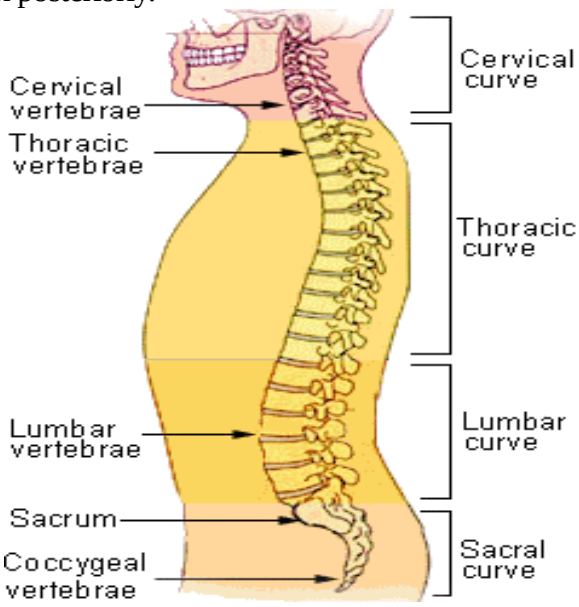


FIGURE 1: ANATOMY OF VERTEBRAL COLUMN

A typical vertebra has two parts:

1. anteriorly, BODY or the base which bears the weight.

2. The ARCH surrounds the cord laterally and posteriorly consists of lamina and

pedicles.

3. There are seven processes or projections:

a. Three muscular processes - two transverse and one spinous.

b. There are four articular processes - two upper and two lower.

The typical thoracic vertebra:

It has a heart-shaped body bearing one or two facets for articulating with the head of a rib. Its vertebral foramen is smaller and circular than those of the cervical and lumbar regions.

The two pedicles bear long and strong transverse processes. It articulates with the neighbouring vertebra by means of articular processes that bear vertical facets superior facing posteriorly and inferior facing anteriorly. Its spinous process is long and slopes posteroinferiorly so that the tip overlies the level of the vertebral body below.

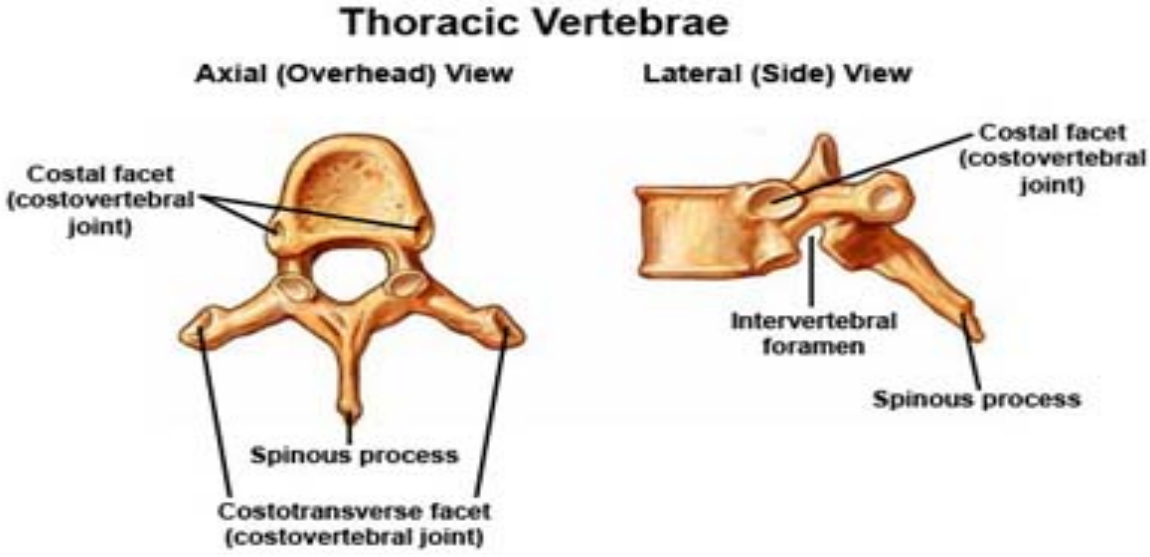


FIGURE 2: THORACIC VERTEBRA

The typical lumbar vertebra

It has a larger kidney-shaped body and its vertebral foramen is larger when compared to that of thoracic vertebra. Its transverse processes are long and slender. Its articular processes are directed (superior) posteromedially and (inferior) anterolaterally. Its spinous processes are shorter, broader and more horizontal than those of thoracic vertebrae.

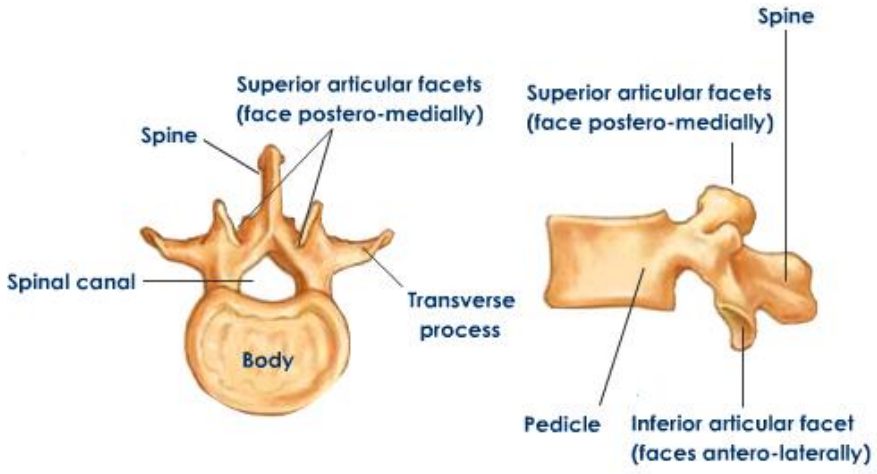


FIGURE 3: LUMBAR VERTEBRA

Intervertebral disc

These are the connecting links between the vertebral bodies and they account 25% of the length of spine. Each disc adheres above and below to the hyaline cartilage which covers the facet of adjacent vertebral body in front and behind and the disc also gets attached to the anterior and posterior longitudinal ligaments.

Joints of the vertebral column

The vertebrae articulate at the intervertebral and facet joints. The intervertebral joints are located between adjacent vertebral bodies. They maintain the strength of

attachment between vertebrae. The facet joints lie between superior and inferior articular processes.

Contents of vertebral canal:

1. Spinal cord and meninges.
2. Cerebrospinal fluid.
3. Spinal nerve roots.
4. Vessels, areolar tissue.

SPINAL CORD:

Medulla oblongata continues as spinal cord below the level of foramen magnum tapering off into conical extremity known as conus medullaris. From the apex of conus medullaris, delicate fibrous filaments descend into the back of first segment of coccyx, which is called as filum terminale. At birth, the spinal cord ends at the level of lower border of third lumbar vertebra. In adults the level of termination of spinal cord may be as follows:

• Lower border of L1 vertebra 50%
• Upper border of L2 vertebra 40%
• Lower border of L3 vertebra 10%

THE MENINGES

Dura mater: It is the outer most and thickest meningeal tissue. It begins at the foramen magnum, where it fuses with the periosteum of the skull. The dural sac of spinal dura mater is the continuation of the cranial dura mater. It is a circular sac or sleeve surrounding the spinal cord. Caudally it ends at S2 where it fuses with filum

terminale. It extends laterally along the spinal nerve roots to become continuous with the connective tissues of the epineurium at the level of intervertebral foramina. The dura mater is composed of randomly arranged collagen fibres along with longitudinally and circumferentially arranged elastin fibres. It is largely acellular except for a single layer of cells that forms the border between the dura and arachnoid mater. Despite the lack of cellular elements, the inner edge is highly vascular, due to which it forms an important route for drug clearance from epidural and subarachnoid space. The inner surface of the dura mater abuts the arachnoid mater. There is a potential space between these two membranes called subdural space. Occasionally it is possible to inadvertently insert an epidural catheter into the subdural space.

Lower extent of dural sac can be as follows:

S_2 vertebra -35%

Below S_2- 40%

Above S_2- 25%

Below this the dura continues as the covering of filum terminale.

Arachnoid mater: It is a delicate, avascular membrane composed of overlapping layers of flattened cells with connective tissue fibres running between the cellular layers. These cells are interconnected by frequent tight junctions and occluding junctions, which account for the fact that arachnoid mater acts as the main barrier for drugs moving between the epidural space and spinal cord. Superiorly the spinal arachnoid continues with the cranial arachnoid and inferiorly it ends along with dura at the lower border of S_2. Arachnoidmater herniates through the dura into the epidural space near the spinal nerve roots forming arachnoid granulations. These granulations

serve as exit for the drugs from subarachnoid space. The subarachnoid space lies between arachnoid and pia mater and contains CSF.It is in continuity with cranial CSF and provides route for the drugs to reach brain.

Pia mater: It is also a delicate, highly vascular membrane which adheres intimately to the spinal cord and dips into the long sulci along its surface. It consists of a thin layer of connective tissue cells interposed with collagen. There are trabeculae which connect the piamater with arachnoid mater and the cells of these two meninges blend together along the trabeculae.Fenestrations in pia mater help in communication with the subarachnoid space. Pia forms a separate sheath for each nerve rootlet as far as the intervertebral foramina, where it binds with the epineurium. At the lower end of the cord, pia continues as a thin thread the filum terminale which becomes invested by the dural (external) filum terminale and continues to the coccyx. Considered an extension of piamater, the delicate ligament is much tougher and less vascular than that of pia. These are attached to the dura by a series of 20-21 meticulous processes (tooth like projections) which aid in the supporting the spinal cord.

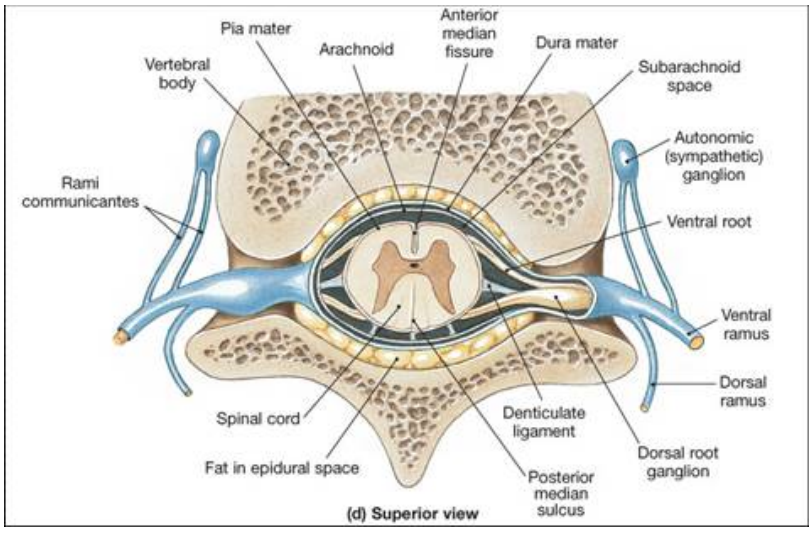


FIGURE 4: MENINGES

Spinal segments

The spinal cord is divided into segments by means of spinal nerves arising from it. The spinal nerves are 31 in number and are as follows:-

8 Cervical

12 Thoracic

5 Lumbar

5 Sacral

1 Coccygeal (rudimentary)

The nerve roots within the dura have no epidural sheath. Therefore they are easily affected by doses of analgesic drugs brought into contact with them.

Spinal nerves

Two roots, anterior and posterior fuse together forming spinal nerves. Sympathetic preganglionic fibres arise from cells in the intermediolateral horn of the spinal cord from T1 to L2. The posterior root is larger than the anterior and the efferent impulses from whole body including viscera pass through these roots. Each posterior root has a ganglion which conveys afferent impulses of touch, pain, temperature, deep sensation from joints, muscles and tendons as well as afferent impulses from viscera.

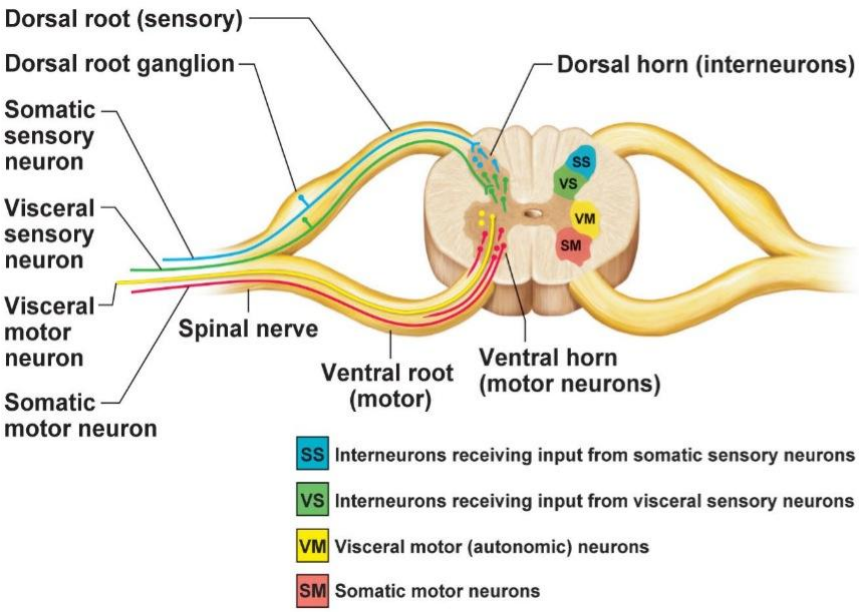


FIGURE 5: CROSS SECTION OF SPINAL NERVE

Segmental levels

1. Perineum S1- S4
2. Inguinal region L1
3. Umbilicus T10
4. Subcostal T6-9
5. Nipple line T4-5
6. Second intercostal space T2
7. Clavicle C3-4

The skin above the nipple has double innervations from C3-4 and T 2, 3, 4. So even with a successful block to C8 there will be some sensation above the nipple line.

Blood supply of spinal cord

The principle arterial supply of the spinal cord is derived from one anterior and two posterior spinal arteries which descend from the level of foramen magnum. The anterior spinal artery is formed by the union of branches from each vertebral artery at the foramen magnum. It supplies anterior 2/3 of the spinal cord. There are two posterior spinal arteries, one on each side. They are derived either directly from the vertebral artery or more often from a primary branch of each vertebral artery. They supply the posterior 1/3rd of the spinal cord. This supply is augmented by spinal branches of the vertebral, ascending cervical, posterior intercostals, lumbar and lateral sacral arteries, which also pass through the intervertebral foramina. Venous drainage is via a plexus of anterior and posterior spinal veins in the neck, the azygous vein in thorax, lumbar veins in the abdomen and lateral sacral veins in the pelvis.

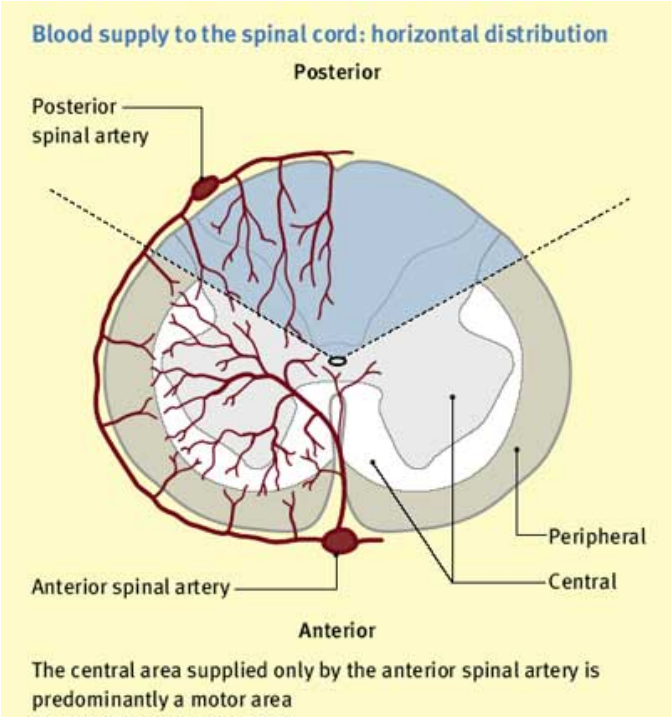


FIGURE 6: BLOOD SUPPLY OF SPINAL CORD

ANATOMY OF EPIDURAL SPACE[16,17]

The epidural space is a circular space surrounding the dura which extends from foramen magnum to the coccyx. The lower limit is sacrococcygeal membrane. The space is potential and exists between the lining of the vertebral canal and the dural sac. Structures encountered from skin to the epidural space by the advancing epidural needle in midline approach are:

1. Skin
2. Subcutaneous tissue
3. Supraspinous ligament
4. Interspinous ligament
5. Ligamentum flavum

Ligaments

a. Supraspinous ligament runs superficial to the spinous processes

b. Posterior spinous processes are connected by Interspinous ligament

c. Laminae are connected by Ligamentum flavum

Supraspinous ligament

It is a strong fibrous cord that connects the apices of the spinous processes from the sacrum to C7, where it continues as ligamentum nuchae. It is thickest and broadest in the lumbar region and varies with age, sex and body built.

Interspinous ligament

It is a thin membranous ligament that connects the spinous processes blending anteriorly with the ligamentum flavum and posteriorly with the supraspinous ligaments. Like supraspinous ligaments, the interspinous ligaments are thickest and broadest in the lumbar region.

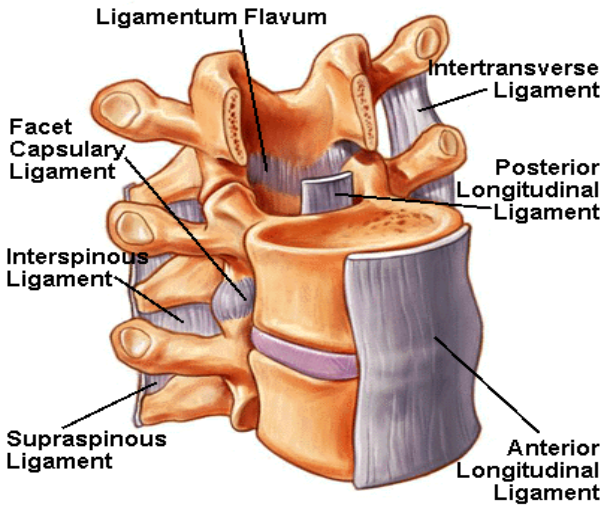


FIGURE7:LIGAMENTS

Ligamentum flavum

It comprises of yellow elastic fibers and connects adjacent laminae that run from the caudal edge of the lamina above to the cephalic edge of the lamina below. Laterally, this ligament begins at the roots of the articular processes and extends posteriorly and medially to the point where the laminae join to form the spinous process. Hence the two components of the ligament are limited, thus covering the interlaminar space. Because of its elasticity and its thickness of several millimetres in the lumbar region, the ligaments impart a characteristic 'springy' resistance, particularly to large bore needle with an upturned end [Tuohy needle]. The ligament thickness, distance to dura and skin to dura distance vary with the area of vertebral canal.

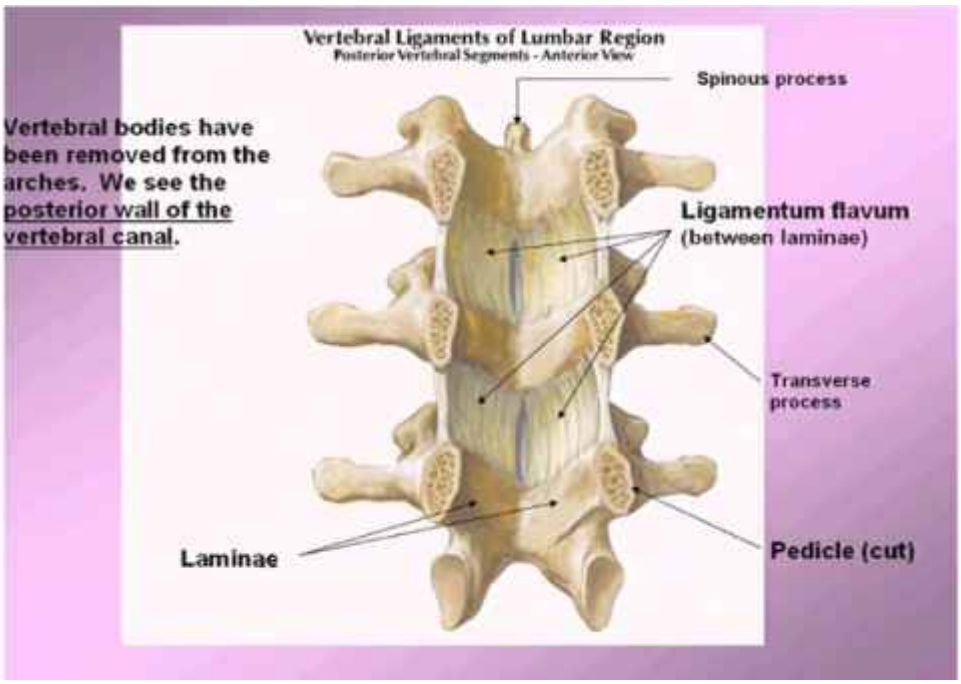


FIGURE 8: LIGAMENTUM FLAVUM

Table: 1 Thickness of ligamentum flavum at different levels[18]

Level	Thickness
Cervical	1-2 mm
Thoracic	3-5 mm
Lumbar	5-6 mm
Caudal	2-6 mm

BOUNDARIES OF THE EPIDURAL SPACE

Superior: The foramen magnum. The two layers of the dura mater are attached to the margins of the foramen magnum.

Inferior: Sacral hiatus and the sacrococcygeal membrane.

Lateral: Periosteum of the pedicles and the inter-vertebral foramina.

Anterior: Posterior longitudinal ligament covers the vertebral bodies and the intervertebral discs.

Posterior: The anterior surfaces of the laminae and their connecting ligaments, roots of the vertebral spines and the ligamentum flavum.

CONTENTS OF THE EPIDURAL SPACE

Dural sac and the spinal nerve roots.

Epidural plexus of veins.

Epidural fat.

Lymphatics.

Spinal arteries.

Epidural fat

It consists of semifluid lobulated areolar tissue. It is most abundant posteriorly, diminishes adjacent to the articular processes, and increases laterally around spinal nerve roots, where it is continuous with the fat surrounding the spinal nerves in the intervertebral foramina and hence with the fat in the paravertebral space. Overall the amount of fat in the epidural space tends to vary in direct relation to that

present elsewhere in the body, so that obese patients may have epidural spaces that are occupied by generous amount of fat. The fat itself has a great affinity for drugs with high lipid solubility, which may remain in epidural fat for longer periods. Uptake of local anaesthetics into epidural fat competes with vascular and neural uptake.

Epidural veins

The large valveless epidural veins are part of the internal vertebral venous plexus, which drains the neural tissue of the cord, the CSF and the bony spinal canal. The major portion of this plexus lies in the anterolateral part of the epidural space. The plexus has rich segmental connections at all levels within the intervertebral foramina, epidural space and within the body of the vertebrae. Superiorly, the plexus communicates with the occipital, sigmoid and basilar venous sinuses within the cranium. Inferiorly, anastomosis by way of the sacral venous plexus links the vertebral plexus to uterine and iliac veins.

By way of intervertebral foramina at each level, the vertebral plexus communicates with the thoracic and abdominal veins, so that pressure changes in these cavities are transmitted to epidural veins but not to the supporting bony elements of the neural arch and vertebral bodies. Thus, marked increase in intra abdominal pressure may compress the inferior vena cava while distending the epidural veins, increasing flow upto the vertebrobasilar plexus. This increased flow is accommodated mostly by means of the azygous vein, which ascends in the right chest over the root of right lung into the superior vena cava. Distension of epidural veins, owing to direct inferior vena cava obstruction [as in pregnancy] or owing to

increased thoracic and abdominal pressure, will also diminish the effective volume of the epidural space, with the result that injected local anaesthetic spread more widely up and down the epidural space.

Three important aspects of safety Include:

1. The epidural needle should pierce the ligamentum flavum in the midline to avoid the laterally placed epidural veins.

2. Insertion of epidural needles or catheters or injections of local anaesthetics should be avoided during episodes of marked increase in size of epidural veins, such as during straining in labour.

3. The presence of venacaval obstruction calls for a reduction in dose, a decreased rate of injection and increased care in aspirating blood before epidural injection.

Spinal arteries

It is of significance to epidural block that the spinal branches of the subclavian, aortic and iliac arteries enters the epidural space in the region of the dural cuffs. The anterior spinal artery territory supplying the anterior horn or motor area of the spinal cord is the most vulnerable as it is a single artery and does not anastomose with the two posterior spinal arteries.

Epidural lymphatics

The dural cuff region is supplied with rich lymphatic network that rapidly conveys debris from arachnoid villi out through intervertebral foramina to reach lymph channels in front of the vertebral bodies.

Dural sac

Encases dura, arachnoid, spinal fluid, pia, spinal nerves and spinal cord.

Epidural pressure

In the lumbar region, the major cause of generation of a negative pressure lies in coning of the dura by the advancing needle point. Negative pressure increases as the needle advances across the epidural space towards the dura. Blunt needles with side openings produce the greatest negative pressure; they produce a good coning effect on the dura without puncturing it and transmit the negative pressure well because of their side opening.

Slow introduction of the needle produces the greatest negative pressure. Greatest negative pressure can be obtained if the dura is not distended [eg. By gravity in sitting position or by high abdominal or thoracic pressure]. In pregnancy, the epidural space may well have a positive pressure. Hence hanging drop technique may not be reliable in pregnant women to identify the epidural space.

Table: 2 Thickness of epidural space at different levels[18]

Levels	Thickness(mm)
Cervical	2-3
Thoracic	3-5
Lumbar	5-6
Sacral	2-6

HISTORY OF EPIDURAL ANAESTHESIA

History of epidural and intrathecal anaesthesia and analgesia has been in parallel with the development of general anaesthesia. As ether anaesthesia (1846) is considered the first modern anaesthetic since its use by Morton 162 years ago, so also was August Bier, the first to introduce intrathecal anaesthesia by using cocaine in 1898. The discovery of the local analgesic effect of cocaine by Carl Koller in 1884 made possible the vast array of regional analgesic therapy.[19]

Interspinous approach to the epidural space was first introduced by Pages in 1921[20] and further popularized by Dogliotti in 1951[21].Next important development was the adaptation of Tuohy's catheter technique in 1945 for continuous spinal anaesthesia by Tuohy[22] and epidural anaesthesia by Curbello[23] in 1949.Advantages of epidural anaesthesia is that a single injection will provide perioperative analgesia, muscular relaxation, graded hypotension and decreased blood loss.

The first epidural injection was introduced by Jean Athanase Sicard, a French neurologist and Cathelin FC in 1901.Sicard also described "loss of resistance"technique for locating the epidural space in 1921.Lumbar epidural technique was first performed by Fidel Pages of Spain in1921 for abdominal surgeries.[20]

A.M.Dogliotti,[21] an Italian surgeon has been considered the father of modern epidural anaesthesia. He described in detail the anatomy and physiology of epidural space and developed the modern "loss of resistance"technique for clinical location of epidural space [18] in 1932. Gutierrez taking advantage of the negative pressure in epidural space described the "sign of drop".

EPIDURAL NEEDLES

In 1944, Tuohy used 15 G Barker needle through which no.4 ureteric silk catheter was passed into subarachnoid space.[22].A Seattle dentist Huber RL, invented hypodermic needle with a long, sharp and curved tip to lessen the pain on injection. Tuohy recognised that this curved tip (Huber point) would facilitate placement of epidural catheter and applied this design to his needle in 1945.He also added a stylet to decrease the risk of skin plugging.[22,24]

In 1954 Hustead introduced epidural needle with a rounded heel to reduce the danger of trapping the catheter. Weiss was practising hanging drop method for locating epidural space. He introduced metal wings to the hub of the epidural needle. In 1987, Sprotte introduced pencil point epidural needle, to minimise tissue trauma.[25]

EPIDURAL CATHETERS

The first indwelling catheter to be used for continuous epidural anaesthesia was silk 3.5 to 4F ureteric catheters. Flowers in 1949 described the use of plastic catheters. First material to be used was polyethylene, which was replaced by polyvinylchloride (PVC).Recently nylon, Teflon, polyurethane and silicone materials are being used to produce thin, yet kink resistant catheter with good stiffness and tensile strength.

PHYSIOLOGY OF EPIDURAL ANAESTHESIA [1]

Originally negative extradural pressure was described in 1928 by Heldt and Moloney. This so called negative pressure in the peridural space is greatest at points of firm attachments. It is also greatest in the thoracic region, less in the lumbar region and least in the sacral area.

Possible causes of negative pressure in the epidural space include:

1. CONE THEORY: Dimpling / coning of dura by the advancing needle.

2.TRANSMISSION THEORY: Transfer of negative pressure from thorax (transmission of negative intrapleural pressure into the peridural space).

3. The initial bulge of the yellow ligament, in front of the advancing needle followed by its rapid return to the resting position once the needle has perforated the ligament.

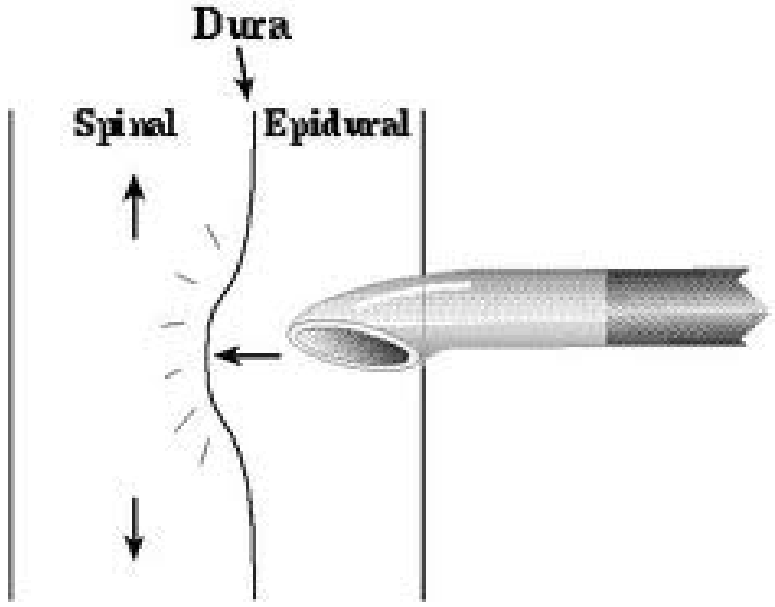


FIGURE 9: NEGATIVE PRESSURE IN EPIDURAL SPACE

Identification of Epidural space

Loss of resistance method:

1. Syringe technique
2. Spring loaded technique
3. Balloon technique (Macintosh's epidural space indicator)
4. Brooks device
5. Vertical tube of Dawkin

Negative pressure technique

1. Hanging drop sign
2. Capillary tube method
3. Manometer technique

Others

1) Ultrasonic localization

2) Oxford epidural space indicator

Newer techniques

1) Auditory amplification method

2) Doppler guidance

3) Pressure transducer guided method

Factors affecting epidural anaesthesia

1. Site of injection

After lumbar injection, analgesia spreads to a greater extent, cranially, with a delay at the L5 and S1 segments, due to larger size of these nerve roots.

After thoracic injection, analgesia spreads evenly from the site of injection. The upper thoracic and lower cervical roots are resistant to blockade due to their larger size. The epidural space in the thoracic region is usually smaller and a lower volume of local anaesthetic is needed.

2. Age, height & weight

There is an age related decrease in the size and compliance of the epidural space due to which there is a decrease in the volume of local anaesthetic needed to achieve a given level of block. The patient's height correlates to some extent with the volume of local anaesthetic needed, so that shorter individuals need lower volume of local anaesthetic i.e. 1 ml per segment to be blocked, while volumes up to 2 ml per segment may be required in case of taller individuals. The safest approach is to inject incremental doses and monitor the effect carefully.There is little correlation between the weight of the patient and the volume of local anaesthetic needed, but in morbidly obese patients the epidural space may be narrowed due to the increase in intra-abdominal pressure, and hence a smaller volume of local anaesthetic is needed.

Furthermore, epidural venous engorgement due to compression of the azygous venous system may further reduce the size of the epidural space, and increase the risk of puncture of an epidural vein. The same applies to patients with large intra-abdominal tumours, ascites, and in pregnancy after 28 weeks.

3. Dosage

The dosage required for analgesia or anaesthesia is determined by several factors but usually, 1-2ml of local anaesthetic is needed per segment to be blocked. The spread of local anaesthetic inside the epidural space is unpredictable as its size is variable, as is the amount of local anaesthetic that leaks into the paravertebral space. The dosage is a function of the volume injected and the concentration of the solution used and so the response need not be the same if similar dose is used in a different volume and concentration. A higher volume of local anaesthetic with low concentration will result in a large number of segments to be blocked but with less dense sensory and motor blockade.

It is important to remember that sympathetic fibres having the smallest diameter are the most easily blocked. Even with low concentrations of local anaesthetic the degree of sympathetic blockade is related to the number of segments blocked.

With an epidural catheter, slow incremental dosing is possible which is important in preventing excessively high sympathetic blockade associated with hypotension. The need for "top-up" doses of local anaesthetic is dependent on the duration of action of the drug. Repeat doses should be given before the block could regress to the extent so that the patient does not experience pain. A useful concept is the "time to two-segment regression". This is the time taken from injection of the first dose of local anaesthetic till the time when the maximum sensory level has receded by two segments. When two segment regression has occurred, one half of the original dose should be injected to maintain the block. The time taken for two-segment regression in case of lignocaine is 90-150 minutes, and for bupivacaine it is 200-260 minutes.

4. Vasoconstrictors

• Although the addition of vasoconstrictors to local anaesthetics prolongs anaesthesia with other regional techniques, their effects on epidural anaesthesia is less consistent.

• With bupivacaine, the addition of epinephrine has not been shown to prolong anaesthesia while with lignocaine the addition of adrenaline (usually 1:200000) prolongs the duration of action of lignocaine.

• However, vasoconstriction does reduce the amount of systemic absorption of local anaesthetics, and reduces the risk of toxicity.

5. Posture

The effect of gravity during placement of the epidural technique has an effect on the spread of local anaesthetic with the caudad area getting blocked with more intensity.

6. Alkalinisation of local anaesthetics

Commercially available solutions of local anaesthetics have a pH between 3.5 and 5.5 for chemical stability and bacteriostasis. Most local anaesthetics are weak bases and exist in ionised (hydrophilic) form at this pH. Since neural blockade depends on the penetration of the drug into lipid nerve cell membranes, the non-ionised (lipophilic) form of the drug crosses the lipid membranes more easily. It follows that by increasing the pH of the solution, the proportion of drug in the non-ionised form increases and thus neural penetration is enhanced which in turn speeds up the onset of blockade. The addition of 8.4% sodium bicarbonate (0.5ml per 10ml of local anaesthetic solution) helps to achieve more rapid onset of blockade.

7. Number and frequency of local anaesthetic injections

Whether augmentation or diminution of neural blockade occurs after repeated epidural injection of local anaesthetics depends on the local anaesthetic agent, the number of injections and timing between injections. Tachyphylaxis has been most clearly demonstrated in association with continuous epidural block in patients in whom repeated injections of the short acting amides - lidocaine, prilocaine or mepivacaine are used. The mechanism of tachyphylaxis is not known. It may be partly explained by pH changes in spinal fluid with repeated injections.

PHYSIOLOGICAL EFFECTS OF EPIDURAL BLOCKADE[26]

Epidural blockade implies sympathetic blockade accompanied by somatic blockade, which may involve sensory and motor blockade alone or in combination. Some of the most important physiological effects of epidural blockade can be discussed in relation to either sympathetic blockade of vasoconstrictor fibers below T4 and or of cardiac sympathetic fibers.

Zone of differential blockade

Sensory

In intradural block sympathetic fibers are blocked two or three segments higher than sensory fibers. In extradural block, the relationship is complex. Level of sympathetic block is the same as (or lower than) sensory with epidural blockade. Sympathetic block will be greater when more concentrated solutions are used or when Adrenaline is added..

Motor

In intradural block, the difference between sensory and motor block is two segments. In extradural block, the difference in levels is greater, depending very much on the nature of local analgesic solution. All types of nerve fibers are affected by local anaesthetics, but there is tendency for small, slower conducting fibers to be more readily blocked than large, fast conducting fibers. Between fiber types however, these rules do not hold good. Myelinated preganglionic B fibers which have a faster conduction time are about three times more sensitive to local anaesthetics than the

slower non- myelinated post ganglionic C fibers.

Sensory Aβ fibers appear to be more sensitive to blockade than motor Aα fibers, although of the same conduction velocity, this may be because sensory fibers conduct at a higher frequency. It has been suggested that this selectivity for sensory fibers exhibited by Bupivacaine and Ropivacaine is a function of frequency dependent block, a property not shared by Etidocaine and Amethocaine.

Cardiovascular System

These are the different ways in which extradural block can influence the cardiovascular system. .

1.Vasodilatation of resistance and capacitance vessels. Block of cardiac efferent sympathetic fibers from T1 to T4 resulting in loss of chronotropic and Inotropic drive and fall in cardiac output.

2. The arterial or Bainbridge reflex causing-bradycardia.

3. The operation of Marey's law causing tachycardia.

4. Depression of vascular smooth muscle and β adrenergic blockade of myocardium with fall in cardiac output.

5. Adrenaline effect (if used) following absorption, results in β stimulation and associated rise in cardiac output and reduction in peripheral resistance. Corrective measures may be considered if arterial pressure falls more than 1/3 below its pre-operative level. Slowing of heart rate is caused if any of the anterior roots carrying sympathetic cardiac accelerator fibers are blocked, as may happen in higher spinal blockade above T4, T5. A further cause of slow pulse rate is the lowering of blood pressure in the right atrium consequent to diminished venous return [Bainbridge

(1874-1921) effect].

On the other hand, Tachycardia during spinal analgesia may result from the operation of Marey's Law (a pulse of low tension is fast). Bradycardia is the more frequent effect.

Theories of causation of fall in blood pressure

1. Diminished cardiac output consequent to reduction of venous return to heart, and lack of muscular propulsive force on veins.
2. Dilatation of post arteriolar capillaries and small venules due to paralysis of Vasoconstrictor fibres. Compensatory vasoconstriction takes place in areas not anaesthetized via carotid sinus reflexes.
3. Paralysis of sympathetic nerve supply to heart T1-T4. Bradycardia may give rise to fall in cardiac output.
4. Paralysis of sympathetic nerve supply to the adrenal glands, with consequent catecholamine depletion
5. Absorption of drug into circulation. This is more likely to be a cause of hypotension after extradural than after intradural analgesia because of the large amount of analgesic drug injected.
6. Ischemia and hypoxia of vital centers
7. Hypovolemia, if present, may give rise to fall in blood pressure if central neural blockade is employed.
8. Compression of great vessels within abdomen, by the pregnant uterus, abdominal tumours or abdominal packs may cause severe hypotension in presence of central neural blockade.

Respiratory system

The phrenic nerve supplying diaphragm arises from the anterior roots of C3, C4, C5 and should not be encroached on in spinal anaesthesia, but phrenic nerve paralysis can occur.

Apnoea may be due to medullary ischemia or due to toxic effect of the drug in extradural blocks. During spinal analgesia breathing becomes quite and tranquil. This is not only due to motor blockade, but also due to reduction of sensory input to respiratory centre. Lowered arterial and venous tone also lessens the work of heart and tends to relieve any pre existing pulmonary congestion. The ventilation perfusion during extradural block is not greatly altered and effects on respiratory functions are relatively small with no evidence of changes in FRC or V/Q ratio. The pulmonary gas exchange is preserved.

Vital capacity and forced expiratory volume may be reduced, especially in cigarette smokers. Intercostal muscle paralysis is compensated for by descent of diaphragm, which is made easier by the lax abdominal walls. This is not accompanied by hypoxia and hypercapnia although the ability to cough forcibly to expel secretion is impaired. The patient may become apnoeic so that tracheal intubation is required. Causes may be:

- Inadequate medullary blood flow due to inadequate cardiac output-a serious situation demanding immediate cardiorespiratory support.
- Total spinal anaesthesia with denervation of all respiratory muscles. True phrenic nerve paralysis is uncommon because all motor nerve roots are large and the anaesthetic solution is likely to be weak when it reaches the cervical

region.

- Accidental subdural injection
- Toxic effects of local analgesic drug.
- Injecting narcotic analgesic drugs

Gastrointestinal system

Preganglionic sympathetic fibers from T5 to L1 are inhibitory to gut. There is no effect on oesophagus, the innervation of which is vagus. The small gut is contracted as the sympathetic inhibitory impulses are removed, the vagus being all powerful, sphincters are relaxed and peristalsis is active. Pressure within the bowel lumen is increased.

Nausea and vomiting due to hypotension may occur and usually come on in waves-lasting a minute or so and then passing away spontaneously. Stimuli arising in the upper abdomen may ascend along the unblocked vagi and perhaps the phrenic nerve, and cause discomfort, if the patient is conscious. Infiltration of local anesthetic solutions may prevent this by blocking vagal afferents. Colonic blood supply and oxygen availability are increased, perhaps an important factor in the prevention of anastomotic breakdown following gut resection.

Theories of causation of nausea and vomiting:

1. Hypotension-correction using a pressor drug may relieve nausea

2. Increased peristalsis

3. Traction on nerve endings and plexuses, especially via vagus

4. Presence of bile in stomach due to relaxation of pyloric and bile-duct sphincters

5. Narcotic analgesics (premedication)

6. Psychological factors

7. Hypoxia

Gastric emptying time is quicker when extradural block is employed for postoperative pain relief than when narcotic analgesics are used.

Liver

There are no specific effects of significance. The degree of hypotension that compromises liver function is not known. Liver disease may interfere with the metabolism of local anaesthetic drugs.

Endocrine system

The usual increase of anti diuretic hormone (ADH) during surgery is suppressed. Spinal block delays adrenal response to trauma, whereas surgeries under general anaesthesia cause a rise in steroids. In any case, either regional or general, there is no difference in the postoperative period once the effect of the block wears off. Spinal block suppresses the hyperglycemic response to surgery and stress and so is useful in diabetic patients but this does not extend into postoperative period. The response to insulin is augmented and anaesthetist should be aware of the possibility of hypoglycemia.

Extradural block prevents lymphopenia and granulocytosis after surgery, thus inhibiting the metabolic endocrine response to surgery and thereby preventing immune suppression.

Genito urinary system

Sympathetic supply of kidney is from T11 to L1 via the lowest splanchnic nerves. Any effects on renal function are due to hypotension. Auto regulation of renal blood flow is impaired if mean arterial pressure falls below 50 mmHg. These changes are transient and disappear when blood pressure rises again.

Sphincters of bladder are not relaxed and tone of ureters is not greatly altered. The penis is often engorged and flaccid due to paralysis of the Nervi erigentes [S2 and S3]. This is a useful positive sign of successful block. Post spinal retention of urine may be moderately prolonged as L2 and L3 contain small autonomic fibers and their paralysis lasts longer than that of the larger sensory and motor fibers.

Body temperature

Vasodilatation favours heat loss. Absence of sweating favors hyperpyrexia in hot environments. Catecholamine secretion is depressed, hence less heat is produced by metabolism.

Extradural space is a temperature sensitive zone, whereas intradural space is not. Cold solutions injected into extradural space may induce shivering

1. Because the large veins act as exchangers.
2. As a result of sensory input.
3. Possibly because of the existence of thermal sensors.

PHARMACOLOGY- LEVOBUPIVACAINE

Classification of Local anaesthetics

Local anaesthetic agents consist of a lipophilic group(usually an aromatic benzene ring) separated from a hydrophilic group(usually a tertiary amine) by an intermediate chain that includes an ester or amide linkage.The amino ester group have an ester link and include cocaine, procaine, chloroprocaine and tetracaine. The amino amides have an amide link between the aromatic head and the intermediate chain and include lignocaine, bupivacaine, dibucaine,mepivacaine, prilocaine, etidocaine, ropivacaine and levobupivacaine. [26]

LEVOBUPIVACAINE [12]

CHEMICAL STRUCTURE

Levobupivacaine [(S)-1-butyl-N-(2,6-dimethylphenyl) piperidine-2-carboxamide] is an amino-amide local anaesthetic drug belonging to the family of n-alkyl substitute pipecoloxylidide. Its chemical formula is C18 H28 N2 O.

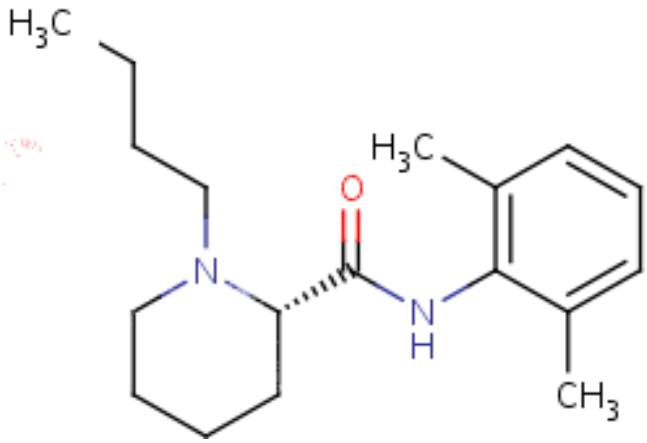


FIGURE 10: CHEMICAL STRUCTURE OF LEVOBUPIVACAINE

PHYSIOCHEMICAL PROPERTIES

Levobupivacaine is a white crystalline powder freely soluble in water and alcohol, slightly soluble in chloroform and acetone. It is commercially available as Chirocaine.

MECHANISM OF ACTION

Levobupivacaine exerts its pharmacological action through reversible blockade of neuronal sodium channels. Myelinated nerves are blocked through exposure at the nodes of Ranvier more readily than unmyelinated nerves; and smaller nerves are blocked more easily than larger ones. In general, the progression of anaesthesia is related to the diameter, myelination and conduction velocity of the affected nerve fibres. Specifically, the drug binds to the intracellular portion(cell membrane) of voltage sensitive sodium channels and blocks sodium influx into nerve cells, which prevents depolarization. It also interferes with impulse transmission and conduction in other tissues.

PHARMACOKINETICS

1. ABSORPTION

The dose as well as the route of administration of levobupivacaine determines the plasma concentration following therapeutic administration as the absorption is dependent upon the vascularity of the tissue. After epidural administration of levobupivacaine, the absorption is biphasic, with rapid absorption of a small quantity of drug into the circulation and slower absorption of the remainder of the drug. It has been observed that peak levels of levobupivacaine in the blood reaches approximately

30 min after epidural administration and doses up to 150 mg had resulted in mean Cmax levels up to 1.2 g/mL. The epidural absorption gets affected by age as the fraction absorbed decreases and the fast absorption phase is shorter in older (aged < 70 years) compared with the younger (aged 18-44 years) patients. The older patients also have a higher spread of analgesia by ~ 3 dermatomes. Therefore, in the elderly patients a lower dose of levobupivacaine, according to their physical status is recommended.

2. DISTRIBUTION: The volume of distribution is estimated at 66.91 ± 18.23 L (after intravenous administration of 40 mg in healthy volunteers). The pKa of levobupivacaine is 8.1, similar to the pKa of the racemic bupivacaine. The half-life is 3.3h. The rate of clearance is 39.06 ± 13.29 L/h (after intravenous administration of 40 mg in healthy volunteers).

Alpha1-glycoprotein is the main binding site for levobupivacaine. Protein binding of levobupivacaine is more (97%) than that of racemic bupivacaine (95%). Less than 3% of the drug circulates free in plasma. The free proportion of the drug can have an action on the other tissues, causing unwanted side-effects and toxic manifestations. In newborns and in protein-deficient states like under nutrition and nephrotic syndrome, lesser amount of protein is available for binding, causing higher levels of free drug, resulting in toxic effects at lower doses.

3. METABOLISM: Levobupivacaine is extensively metabolized with no unchanged levobupivacaine detected in urine or faeces. In vitro studies using (14 C) levobupivacaine showed that cytochrome CYP3A4 isoform and CYP1A2 isoform mediate the metabolism of levobupivacaine to inactive metabolites, desbutyl

levobupivacaine and 3-hydroxy levobupivacaine, respectively. In vivo, the 3-hydroxy levobupivacaine appears to undergo further transformation to glucuronide and sulphate conjugates, which are excreted in urine. Metabolic inversion of levobupivacaine to R (+)-bupivacaine was not evident both in vitro and in vivo.

4. EXCRETION: Following intravenous administration, recovery of the radio-labelled dose of levobupivacaine was essentially quantitative with a mean total of about 95% being recovered in urine and faeces in 48 h. Of this 95%, about 71% was in urine while 24% was in faeces.

Clinical Utility

Levobupivacaine has been increasingly used in clinical anaesthesia practice since last few years because of its safer pharmacological profile. Literary evidence has established the safety of levobupivacaine over bupivacaine when used in regional anaesthesia as the incidence of various adverse outcomes is higher with the latter as compared to levobupivacaine.The low cardiovascular and neurological toxicity of levobupivacaine has led to its use as a local anaesthetic in a wide variety of specialist applications including sub-arachnoid block, epidural anaesthesia and analgesia, brachial plexus blocks, peripheral nerve blocks, ocular blocks as well as local infiltration. It is also being used for intraoperative anaesthesia, labour analgesia, post-operative pain as well as for the management of acute and chronic pain. The introduction of levobupivacaine into Indian market recently has spurred the interest among anaesthesiologists to possibly use it in various clinical situations requiring regional anaesthesia.

Subarachnoid block

Levobupivacaine is an interesting alternative to bupivacaine for spinal anaesthesia. Levobupivacaine produces subarachnoid block with similar sensory and motor characteristics and recovery like bupivacaine. The onset of sensory and motor block is hastened with the use of hyperbaric levobupivacaine as compared to isobaric levobupivacaine. The regression of motor block occurs earlier with levobupivacaine and ropivacaine as compared with bupivacaine. Intrathecal administration of 15 mg of levobupivacaine provides an adequate sensory and motor block lasting for approximately 6.5 h. Smaller doses (i.e., 5-10 mg) are used in day-care surgeries. At low concentrations, levobupivacaine produces a differential neuraxial block with preservation of motor function, which may be favourable for ambulatory surgery. Minimum effective local anaesthetic dose of levobupivacaine as recommended by an up- and-down sequential design study is 11.7 mg.The literary evidence has established that addition of opioids improves the quality of the block with less hemodynamic variations during peri-operative period.

Epidural anaesthesia

Levobupivacaine has been successfully used in providing epidural anaesthesia and analgesia for surgical procedures, which is clearly evident from the summary of various research works.Equal doses of levobupivacaine and bupivacaine (15 mL of 0.5%) provide similar onset of sensory block (8-30 min), maximum cephalic spread (T7-T8) and duration of analgesia (4-6 h). Though, the onset of motor block is delayed with levobupivacaine it is less dense as compared to bupivacaine but with a similar duration.Higher concentration of levobupivacaine (i.e., 0.75% vs. 0.5%)

provides a longer duration of sensory and motor block without any increase in the incidence of adverse side effects. An increase in both volume and concentration of levobupivacaine is however associated with a higher incidence of hypotension (82%) and delayed block regression. The incidence of hypotension is similar when either levobupivacaine or bupivacaine is used for epidural anaesthesia for caesarean section. Levobupivacaine and bupivacaine when used in thoracic epidural anaesthesia provide comparable sensory block and intraoperative hemodynamics as well as similar duration of post-operative analgesia after thoracic surgery.

Post-operative analgesia

Epidural analgesia

A continuous epidural infusion of low concentration of local anaesthetics with or without adjuvants provides excellent post-operative analgesia. Equipotent doses of levobupivacaine, bupivacaine and ropivacaine provide comparable post-operative pain relief and recovery of sensory and motor function. A continuous infusion of 15 mg/h of levobupivacaine provides effective pain relief in the post-operative period. The quality of analgesia is also determined by the concentration of levobupivacaine, i.e., 0.25% solution provides better analgesia as compared to 0.125% or 0.0625% solutions. Levobupivacaine, self-administered via post-operative patient-controlled epidural analgesia also provides good post-operative pain control, similar to ropivacaine, but ambulation occurs earlier in ropivacaine-receiving patients.

The addition of adjunctive agents (epinephrine, opioids or clonidine) to levobupivacaine in epidural anaesthesia and analgesia may provide a dose-sparing effect and increase the duration and quality of analgesia. Epinephrine does not

influence the onset, spread and duration of sensory and motor epidural block or the systemic absorption of levobupivacaine. Clonidine added to levobupivacaine also enhances the quality of analgesia and provides a local anaesthetic sparing effect. The motor block tends to be denser with clonidine and some degree of arterial hypotension occurs.

Wound infiltration

Local anaesthetic infiltration along the incision line is used frequently to provide postoperative analgesia. Post-incisional wound infiltration with 0.125% levobupivacaine provides more effective and longer duration of analgesia and early mobilization as compared to rectal paracetamol, in children after unilateral inguinal hernia surgery. Wound infiltration with levobupivacaine with or without tramadol provides good post-operative analgesia following a caesarean section or lumbar disc surgery. It also increased the inflammatory response and collagen synthesis on both the 8th and 21st days.

Peripheral Nerve Blocks

Different studies have compared levobupivacaine, ropivacaine and bupivacaine in brachial plexus block for upper limb surgeries. Levobupivacaine is a good substitute for bupivacaine. Compared to ropivacaine, levobupivacaine provides a significantly longer duration of analgesia. The return of motor activity is earlier with ropivacaine. The long duration of sensory block associated with good analgesia and less toxicity of levobupivacaine makes it a better choice for upper extremity blocks. Levobupivacaine 0.5% provides a longer duration of sensory block after sciatic nerve block using the Labat approach than the same dose of ropivacaine in foot and ankle

surgery. The use of a single dose of 0.5% levobupivacaine to block the tibial and peroneal nerves for hallux valgus surgery using popliteal approach is preferable over 0.5% ropivacaine for good anaesthesia and better control of post-operative pain. Levobupivacaine 0.5% is as effective as bupivacaine 0.5% and is recommended for the 3-in-1 block.

The quality and duration of peripheral nerve block is improved with the use of higher concentrations of levobupivacaine, (0.5-0.75%).Levobupivacaine administered via a peripheral nerve block continuous catheter provides excellent post-operative analgesia and decreases the post-operative systemic opioid requirements. The addition of adjuvants to the local anesthetics in peripheral nerve blocks such as epinephrine, clonidine or opioids improve the quality of analgesia and provide a dose-sparing effect, thereby decreasing the potential for systemic toxicity. Epinephrine does not add to the inherent long duration of sensory and motor block with levobupivacaine in peripheral nerve blocks but may help to decrease the potential for systemic toxicity. The addition of clonidine and fentanyl to levobupivacaine in paravertebral nerve block provides excellent analgesia and decreases post-operative systemic opioid requirement. Similarly, the addition of tramadol to levobupivacaine in middle interscalene block significantly increases the duration of sensory block.

Obstetric Anaesthesia and Analgesia

Subarachnoid block for caesarean delivery

The time to onset of sensory and motor block as well as the duration of analgesia is slightly longer with intrathecal levobupivacaine as compared to bupivacaine in caesarean section. A potency hierarchy of intrathecal bupivacaine <

levobupivacaine < ropivacaine in caesarean section patients has been confirmed in clinical studies. The accidental intrathecal placement of an epidural-intended catheter can be confirmed with a test dose of 10 mg levobupivacaine.

Labour analgesia

Combined spinal-epidural labour analgesia

Combined spinal-epidural (CSE) technique is widely used in obstetric practice to provide optimal analgesia. It offers effective, rapid-onset analgesia with minimal risk of toxicity. Minimum effective local anaesthetic concentration studies using a CSE analgesia technique (CSE) for labour confirm the potency hierarchy in the following order bupivacaine < levobupivacaine < ropivacaine. The intrathecal minimum local analgesic doses were 2.73-3.16 mg for levobupivacaine and 3.33-3.96 mg for ropivacaine. The addition of fentanyl to levobupivacaine prolongs the duration and increases the success rate of the sensory block after intrathecal administration in a CSE analgesia technique.The addition of epinephrine to a mixture of levobupivacaine and opioid increases the success rate of sensory block, but also increases the frequency of motor blockade.

Ophthalmic Surgery

The low cardiovascular and neurological toxicity of levobupivacaine has led to its application as a preferred local anaesthetic in various ocular blocks including peribulbar block for cataract surgery and retro bulbar block for vitreo-retinal surgery.

At equipotent doses and concentrations, 0.75% levobupivacaine provides more effective peribulbar anaesthesia and more effective post-operative analgesia for

vitreo-retinal surgery compared with 0.75% ropivacaine. Topical anaesthesia with levobupivacaine 0.75% has been found to be more effective than lidocaine 2% in preventing pain and improving patient and surgeon comfort during cataract surgery, with similar toxicity. Levobupivacaine (0.5%) has better anaesthetic properties with respect to 0.75% ropivacaine and is well-suited for peribulbar block in cataract surgery.

Paediatric Anaesthesia

Levobupivacaine is also increasingly being used in pediatric anaesthesia for subarachnoid block, caudal block, epidural anaesthesia and as a continuous epidural infusion for post-operative analgesia.

Subarachnoid block

The dose of levobupivacaine for spinal anaesthesia in neonates is slightly higher than for bupivacaine or ropivacaine. Appropriate doses for infant spinal anaesthesia are 1 mg/kg of isobaric 0.5% bupivacaine and ropivacaine and 1.2 mg/kg of isobaric 0.5% levobupivacaine.

Caudal block

The recommended dose of levobupivacaine for effective caudal anaesthesia has been reported to be 2.5 mg/kg. It appears to be of equivalent potency to racemic bupivacaine in children requiring lower abdominal surgery. Post-operative epidural infusions of 0.125% levobupivacaine or ropivacaine in children produce significantly less motor blockade with equally good analgesia as compared to a similar infusion of bupivacaine.

Geriatric Anaesthesia

Elderly patients coming up for various surgeries including transurethral resection of the prostate or bladder tumour, orthopaedic trauma or joint replacement, cataract surgery, usually have some coexisting cardiac or pulmonary disease. Owing to its safer pharmacological profile, levobupivacaine is considered to be a better local anaesthetic than bupivacaine when used for subarachnoid block in the geriatric population having co-morbid systemic diseases.

ADVERSE DRUG REACTIONS

1. Hypotension

2. Nausea, vomiting

3. Dizziness, headache

4. Tachycardia or bradycardia

5. Effects due to over dosage or unintentional intravascular injection

6. Neurological damage-rare complication.

DRUG INTERACTIONS

Metabolism of levobupivacaine may be prolonged by CYP3A4 inhibitors ketoconazole and CYP1A2 inhibitors methylxanthines.

To be used with caution in patients receiving anti arrhythmic agents with local anaesthetic activity such as mexilitene.

PHARMACOLOGY OF DEXMEDETOMIDINE :[28-31]

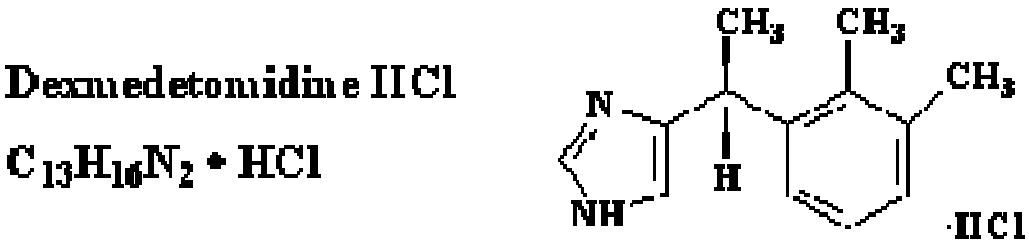


FIGURE 11: CHEMICAL STRUCTURE OF DEXMEDETOMIDINE

Dexmedetomidine is a potent and highly selective alpha 2 adrenoreceptor agonist. It exerts analgesic, sedative and anxiolytic effects after intravenous administration. It was introduced as a sedative especially useful when spontaneous breathing is essential. It is a valuable adjunct during surgery because of its anaesthetic and analgesic sparing effects. It has emerged as an alternative to propofol, opioids and benzodiazepines.

MECHANISM OF ACTION :[29, 30]

Highly selective for alpha 2 receptors over alpha 1(1620:1).Alpha 1 receptors are mainly involved with the regulation of vascular tone. Alpha 2 receptors are mainly pre and post synaptic receptors found in peripheral nervous system and central nervous system.Alpha 2A receptors are mainly located in locus ceruleus and are responsible for sedation,anxiolysis and sympatholysis mediated by G protein inhibition of L type calcium channels. The effects of dexmedetomidine are subcortical and do not cause impairment of cognitive function.

Dexmedetomidine produces analgesic effect by action on alpha 2 receptors in locus ceruleus and spinal cord. Stimulation of alpha 2 receptors reduces central sympathetic output, results in increased firing of inhibitory neurons and alpha 2 receptors in the dorsal horn of spinal cord modulates release of substance p to produce analgesic effects.

EFFECTS ON CVS

1. Decreased heart rate

2. Decreased systemic vascular resistance

3. Decreased myocardial contractility

4. Decreased cardiac output

5. Decreased systemic blood pressure.

Beneficial effect on myocardial oxygen balance decreases perioperative myocardial ischemia in cardiac as well as non cardiac surgery.

PHARMACOKINETICS :[30, 31]

1. ABSORPTION & DISTRIBUTION

Distribution half life after IV administration is 6 minutes. Volume of distribution is 118 l/kg and protein binding is 94%.Kinetics is linear over the dose range of 0.2 to 0.7 mcg/kg/hr.Dexmedetomidine exhibits linear pharmacokinetics in the dosage range of 0.2 to 0.7 mcg/kg/hr when administered by intravenous infusion for upto 24 hours.

2. METABOLISM

Undergoes complete biotransformation in liver via glucuronidase and cytochrome p450 (CYP2A6)-mediated metabolism.The major metabolic pathways of dexmedetomidine are:direct N-glucuronidation to inactive metabolites; aliphatic hydroxylation(CYP2A6) to generate 3-hydroxy dexmedetomidine,glucuronide of 3 hydroxydexmedetomidine and 3 carboxy dexmedetomidine; N-methylation of dexmedetomidine to generate 3 hydroxy N-methyl-dexmedetomidine,3 carboxy N-methyl dexmedetomidine,and dexmedetomidine N-methyl O-glucuronide.

3. EXCRETION

Metabolites are eliminated via kidneys in urine (95%) and in faeces (4%) Elimination half life is 2 hours. Clearance rate 39 l/hr.

DOSAGE: 1 Microgram/kg followed by 0.2 to 0.7 microgram/kg/hr.

CLINICAL APPLICATIONS

1. Attenuates hemodynamic response to laryngoscopy and intubation and reduces the dose requirement of other anaesthetic drugs.

2. Improves perioperative hemodynamic stability in laparoscopic surgeries, craniotomies.

3. Reduces myocardial oxygen demand and hence perioperative myocardial ischemia.

4. IV/intrathecal/epidural dexmedetomidine prolongs the action of local anaesthetics. (Sensory, motor blockade and analgesia)

5. Used for induced hypotension in ENT surgeries

6. For ICU and short procedural sedation.

DRUG INTERACTIONS

Administration with other sedatives results in enhanced sedative effects. Although dexmedetomidine undergoes metabolism by CYP450, no drug interactions involving this pathway have been identified.

Concurrent administration of dexmedetomidine and digoxin results in severe bradycardia.

ADVERSE REACTIONS

1. Hypotension, bradycardia, sedation

2. Nausea, vomiting

3. Dry mouth

4. Cardiac arrest

5. Urinary retention

REVIEW OF LITERATURE

Jung SM et al[32] conducted a randomized double blind study to compare the efficacy and safety of epidural anaesthesia produced by levobupivacaine,bupivacaine and ropivacaine for cesarean section.90 parturients undergoing elective caesarean received epidural block with 20 ml of 0.5% bupivcaine(group B),0.5% ropivacaine(group R) or 0.5% levobupivacaine (group L).Onset time to T6 was 15.7+/-9.8 mins in group L,11.1+/-5.9 mins in group B,18.1+/-13.1 mins in group R.Maximal block height achieved was T4 in all three groups.They also concluded that levobupivacaine produced longer duration of sensory block and shorter duration of motor blockade than bupivacaine and ropivacaine.Bupivacaine produced longer duration and higher incidence of Bromage 3 motor block.Degrees of abdominal muscle relaxation were comparable in all groups.

Peduto VA et al[33] compared the onset time and duration of epidural anaesthesia produced by levobupivacaine and ropivacaine.ASA PS I-III adults undergoing elective lower limb procedures were randomized to receive epidural levobupivacaine 0.5% 15 ml or epidural ropivacaine 0.75% 15 ml.With levobupivacaine onset time was 29+/-24 min and with ropivacaine it was 25+/-22 min.Complete resolution of motor block was 105+/63 min with levobupivacaine and 95+/-48 min ropivacaine.

Bader AM et al[34] compared the efficacy of 0.5% levobupivacaine with 0.5% bupivacaine for epidural anaesthesia in 60 parturients undergoing elective caesarean delivery.patients randomly received 30 ml of either 0.5%levobupivacaine or 0.5% bupivacaine.Levels of sensory block,motor block,muscle relaxation and overall quality of anaesthesia did not differ between groups.The incidence of hypotension was 84.4% in the levobupivacaine group and 100% in the bupivacaine group.

Cox CR et al[35] compared the clinical efficacy and safety of levobupivacaine with bupivacaine for epidural anaesthesia.88 patients undergoing elective lower limb surgery under lumbar epidural anaesthesia received 15 ml of 0.5% levobupivacaine or 0.5% bupivacaine in a double blind manner.There was no difference in onset time,maximum spread of sensory block or intensity of motor block between the two groups.

A randomized double blind study conducted by Tanakka & Ogleari et al[36] included 87 patients undergoing lower abdominal procedures in epidural anaesthesia. Group 1 given 0.5% levobupivacaine, group 2 given 50% enantiomeric excess 0.5% bupivacaine and group 3 given 0.5% bupivacaine. It was concluded that levobupivacaine produced less motor block as compared to other compounds.

In a study conducted by Cox et al 1998[35] ,onset of sensory block ranged between 8 to 30 minutes with maximum upper spread T7-T8 after L2-L3 or L3-L4 lumbar epidural injection and duration 4-6 hours are similar after equal doses of levobupivacaine and bupivacaine(15 ml of 0.5%)

A study conducted by Kopacz et al 2000[37] found that the onset of motor block is slower with levobupivacaine and the quality of motor blockade follows the rank of order bupivacaine>levobupivacaine>ropivacaine. However increasing both volume and concentration of levobupivacaine prolong the duration of both sensory and motor blockade which is associated with high incidence of hypotension(82%) and delayed block regression.

As reported by Casati et al [38] a better way to control the quality and duration of epidural block with levobupivacaine without any hemodynamic consequences is via continuous epidural infusion. It was also shown that a continuous epidural infusion is

associated with excellent postoperative analgesia and similar recovery of sensory and motor function after equipotent doses of levobupivacaine, bupivacaine and ropivacaine.

A study conducted by Murdoch et al(2002)[39]compared the continuous epidural infusion of 0.0625% levobupivacaine with 0.125% levobupivacaine at 6 ml/hr for hip & knee replacement surgeries and found that the effective dose of epidural levobupivacaine for continuous postoperative analgesia approached to 15 mg/hr.

Dernedde et al 2006[40] in their study found that the use of large concentration, small volume of epidural infusion (i.e) 3ml/hr of levobupivacaine 0.5% provides better analgesia with significant hemodynamic stability

Wang LZ and Chang XY et al[41] compared bupivacaine,ropivacaine and levobupivacaine with sufentanil for patient controlled epidural analgesia during labour and concluded that equipotent low concentrations of levobupivacaine, bupivacaine and ropivacaine, all with the addition of sufentanil 10 mcg, produce similar pain relief and motor block, but levobupivacaine and ropivacaine produce a longer lasting analgesia. In patient-controlled epidural analgesia, concentrations of <0.1% levobupivacaine, bupivacaine and ropivacaine with sufentanil produce similar analgesia and motor block with safety for labour analgesia. Both levobupivacaine and ropivacaine are being favored in labour analgesia because of less motor block and less toxicity as compared to bupivacaine.They also found that the analgesic efficacy mainly depends on the concentration rather than the type of anaesthetics and at least 0.1% is needed for satisfactory analgesia. They concluded that Levobupivacaine, Ropivacaine and Bupivacaine all confer adequate and safe labour analgesia, with no significant influence on the mode of delivery, duration of labour, or neonatal outcome.

Elhakim M, Abdelhamid D, Abdelfattach H, Magdy H.et.al (2010)[42] conducted study on 50 adult male patients in thoracic surgery with open lung ventilation, where after induction of general anaesthesia group D received epidural dexmedetomidine 1 mcg/kg with bupivacaine 0.5% & group B received epidural bupivacaine 0.5% alone. They found that use of epidural dexmedetomidine decreases the anaesthetic requirements significantly, prevents awareness during anaesthesia and improves intraoperative oxygenation and post-operative analgesia. [44]

Lopez et al[43] conducted a study on 40 patients for surgery on abdomen and lower limbs (ASAI and II) in a single group. They were given epidural dexmedetomidine 1µ/kg plus lidocaine and epinephrine .It was concluded that use of dexmedetomidine reduces the doses of local anaesthetics as it potentiates the effect of both drugs, with consequent reduction of their adverse effects.

Kanazi et al[44] compared in their study the effects of supplementation of epidural 0.5%bupivacaine with 50 micrograms dexmedetomidine epidurally on 60 patients of ASA I & II. They concluded that epidural bupivacaine with dexmedetomidine produced significantly longer sensory and motor blocks with shorter onset of motor block in comparison to epidural bupivacaine alone.

Kamal et al[45] conducted a comparative study on epidural dexmedetomidine vs epidural morphine added to epidural 0.5% levobupivacaine and found that both the additives produced comparable sensory and motor blockade. There was no statistical significance between the two groups as regards the onset of sensory block, time to total regression of motor or sensory block or maximum sensory block level.Time to reach total motor block was significantly shorter in the Morphine group(LM) than the Dexmedetomidine group(LD).There was no statistically significant difference

between the two groups in the degree of muscle relaxation.There was no statistically significant difference as regards nausea vomiting or respiratory depression. However, 5 patients (16.6%) in the LM group suffered from pruritis while no patient suffered from it in the LD group, and 6 patients (20%) suffered from dry mouth in the LD group while no patient suffered from it in the LM group.

METHODOLOGY

PATIENT SELECTION

GROUP A: 30 patients who received 20 ml of epidural 0.5% isobaric Levobupivacaine with 0.5 ml distilled water.

GROUP B: 30 patients who received 20 ml of epidural 0.5% isobaric Levobupivacaine with 0.5 ml of Dexmedetomidine containing 50µg.

INCLUSION CRITERIA

- Adult patients of either sex belonging to the age group 25-45 years weighing between 50-70kg with BMI ranging between 19-24.
- Patients undergoing elective infraumbilical and lower limb surgeries with site of incision below T10 level such as:
  - Inguinal hernia-Hernioplasty/Herniorrhaphy
  - Appendicitis-Open appendicectomy
  - Hydrocele sac eversion
  - Incisional hernia-Anatomical repair/mesh repair
  - Varicose veins-Trendelenberg procedure
  - Varicocele
  - Split skin grafting & flap cover.
- ASA I & ASA II
- Consent from patient
- Duration of surgery not more than 2 hours.

EXCLUSION CRITERIA

- Patients not willing for the study
- Pregnant women
- ASA III & ASA IV
- Patients who are known to be allergic to study drugs
- Patients in sepsis
- Emergency surgeries
- Infection at the site of injection
- Coagulopathy or other bleeding diathesis
- Severe hypovolemia
- Orthopaedic surgeries
- Gynaecological surgeries
- Duration of surgery more than 2 hours

DRUGS USED

- Inj 0.5% Isobaric Levobupivacaine
- Inj Dexmedetomidine
- Inj Ephedrine
- Inj Atropine
- Inj Midazolam

MONITORS USED

- Pulse oximeter, NIBP, ECG,Temperature monitors.

MATERIALS & METHODS

- A randomized controlled double blind study was planned. The study solutions were prepared by an anaesthesiologist not involved in the patient care. Patient and anaesthesiologist who delivered the epidural anaesthesia were blinded to the study solutions.
- In case of any emergencies arising, it was planned to decode the patients immediately and patients would be treated accordingly.
- Sampling was based on statistical record of surgical cases done at KMCH & GRH. According to the operative statistics of the year 2013, around 1200 surgical cases(lower abdominal & lower limb surgeries) have been performed (inclusive of both hospitals) over a period of 6 months.
- So based on the above data, out of 600 cases, 60 patients who fulfilled the inclusion criteria were chosen for the study over a period of 6 months.
- All pre-anaesthetic evaluation of the 60 patients under the inclusion criteria were performed a day before the surgery. All 60 patients, after being taken a written informed valid consent were randomly allocated into the following groups.

Group A (n=30) =patients who received epidural 0.5% isobaric Levobupivacaine 20 ml with 0.5 ml of distilled water.

Group B (n=30) =patients who received epidural 0.5% isobaric Levobupivacaine 20 ml plus 0.5 ml Dexmedetomidine containing 50µg.

- In the operation theatre good peripheral intravenous access was secured using

18 gauge cannula.

- Baseline non invasive blood pressure, ECG, pulse rate and SpO_2 were recorded.
- All patients received Ringer lactate solution 20ml/kg as preloading solution before the block. Intravenous fluids were given as per body weight and operative loss requirement.
- Patients were positioned on their right lateral side and skin over the desired site was infiltrated with 1% lignocaine 2ml.
- L2-L3/L3-L4 interspaces were selected and epidural space identified using 18G Tuohy needles, midline approach, using loss of resistance technique with air.
- After exclusion of blood in the needle with negative aspiration, 2 ml of 0.5% isobaric levobupivacaine was injected to exclude intrathecal placement of the needle.
- After which the epidural catheter was inserted and fixed 5 cm inside. Now patients in group A received 18 ml of 0.5% isobaric Levobupivacaine with 0.5 ml distilled water and group B received 18 ml of 0.5% isobaric Levobupivacaine plus 0.5 ml of Dexmedetomidine containing 50µg epidurally.

PARAMETERS OBSERVED WERE

- Baseline pulse rate, SpO_2 at room air, noninvasive blood pressure were recorded.
- Cardio respiratory parameters were monitored continuously and recordings

were made every 5 minutes until 30 minutes and at 10 minute interval for the first 2 hours and for every hour till 6 hours, thereafter 2 hourly till 12 hours

- Intraoperatively, incidence of bradycardia (heart rate<50beats per minute) was treated with 0.6mg of Injection Atropine i.v and hypotension (systolic blood pressure falling more than 20% from the baseline value) was treated with Injection Ephedrine 6 mg IV.
- Time to sensory block at T10 dermatome is the time interval between the initiation of anaesthesia and the onset of cutaneous analgesia at T10.This was evaluated using midline bilateral pin prick every minute till complete loss of cutaneous sensation at T10 at which point surgery was proceeded.
- Maximum level of sensory block reached and time taken to achieve the same.
- Time of onset of motor block (defined as onset of motor block to Bromage scale 1) and degree of motor blockade was assessed using modified Bromage scale.

  0= No block

  1=Inability to raise extended leg

  2=inability to flex the knee

  3=inability to flex ankle and foot

- Sedation scores were recorded just before the initiation of surgery and every 30 minutes. Level of sedation was assessed using Ramsay 5 point scale.

1=alert and wide awake

2=arousable to verbal commands

3=arousable to gentle tactile stimulation

4=arousable to vigorous shaking

5=unarousable

- Duration of analgesia was recorded as time interval from the completion of administration of anaesthetic agent to the time when the patient complains of pain at the surgical incision site with VAS score >3. Procedure was explained and the patients were taught to assess the onset of pain using the visual analogue scale (VAS). In the visual analogue scale the patients were shown a scale of 10 cm length. Zero end of the scale was taken as 'No pain' and 10 cm mark as 'Maximum pain'. Intensity of pain increases gradually from '0' to '10'. VAS score was assessed hourly from the time of completion of surgery. Patients were instructed to point the onset of pain on the scale when VAS score>3.
- Duration of sensory blockade was noted as time interval from the epidural administration of anaesthetic agent till the regression of sensory level to S1.
- Duration of motor blockade was noted as the time interval from the epidural administration of anaesthetic agent till regression of motor blockade to modified Bromage scale 0.
- During surgical procedure adverse effects like nausea, vomiting, dry mouth, dizziness, headache, respiratory depression, pruritis and shivering were recorded.
- Any postoperative untoward side effects were noted for 48 hours.

❖ STATISTICAL ANALYSIS

Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented as Mean ± SD and results on categorical measurements are presented as percentage (%). Chi-square test has been used to find the significance of study parameters on categorical scale between two groups. Student‘t’ test has been used to determine the significance between the means of two groups. All analyses were two tailed and $p < 0.05$ was considered significant. SPSS version 16.0 was used for data analysis.

OBSERVATION AND RESULTS

Sixty patients of either sex belonging to ASA I and II were selected and randomly divided into two groups. The data collected were analysed and the results were tabulated using the Statistical Package for Social Sciences. Appropriate statistical analysis of data were done using following tests:

1. Student t test for parametric data.

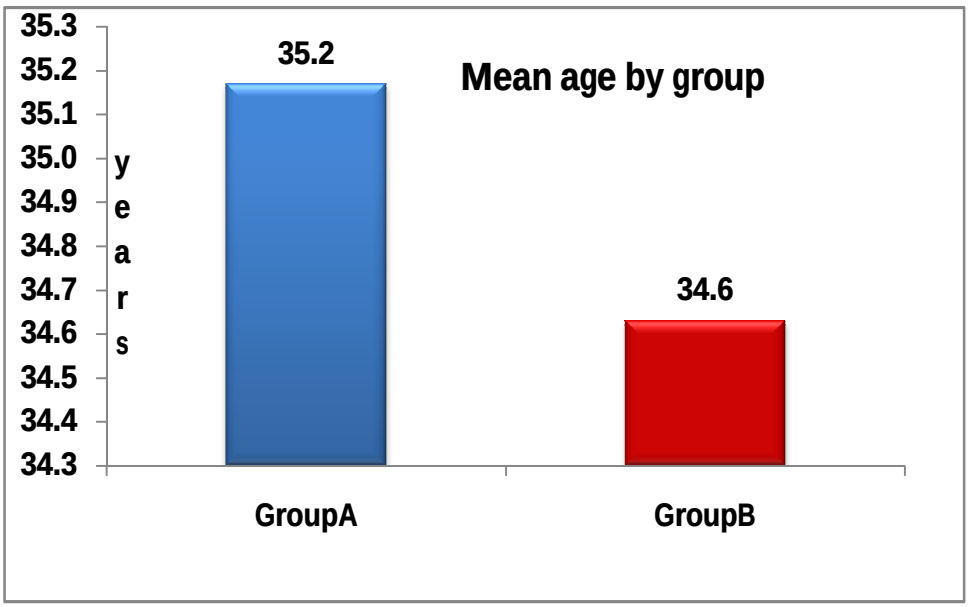
2. Chi-square test for non parametric data.

P<0.05 was considered as statistically significant.

AGE DISTRIBUTION

There was no statistically significant difference in the distribution of age among the two groups, the p value being 0.693.

AGE (IN YEARS)			
GROUP	**MEAN**	**STANDARD DEVIATION**	**p-VALUE**
A	35.17	4.969	0.693
B	34.63	5.147	

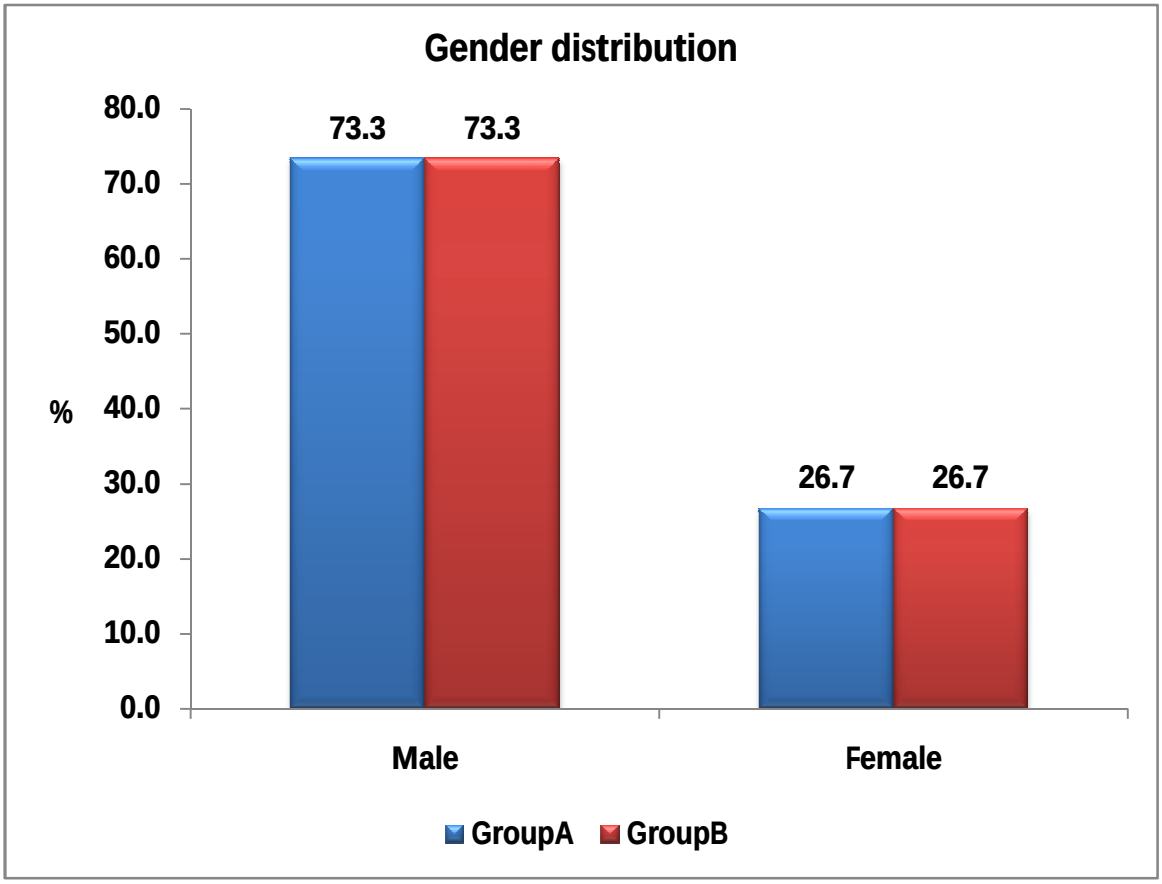


SEX DISTRIBUTION

The distribution of sex between the two groups did not have statistical difference with p value of 1.

	GROUP		SEX	
			Male	Female
	A	Count % within Group	22 73.3%	8 26.7%
	B	Count % within Group	22 73.3%	8 26.7%
Total		Count % within Group	44 73.3%	16 26.7%

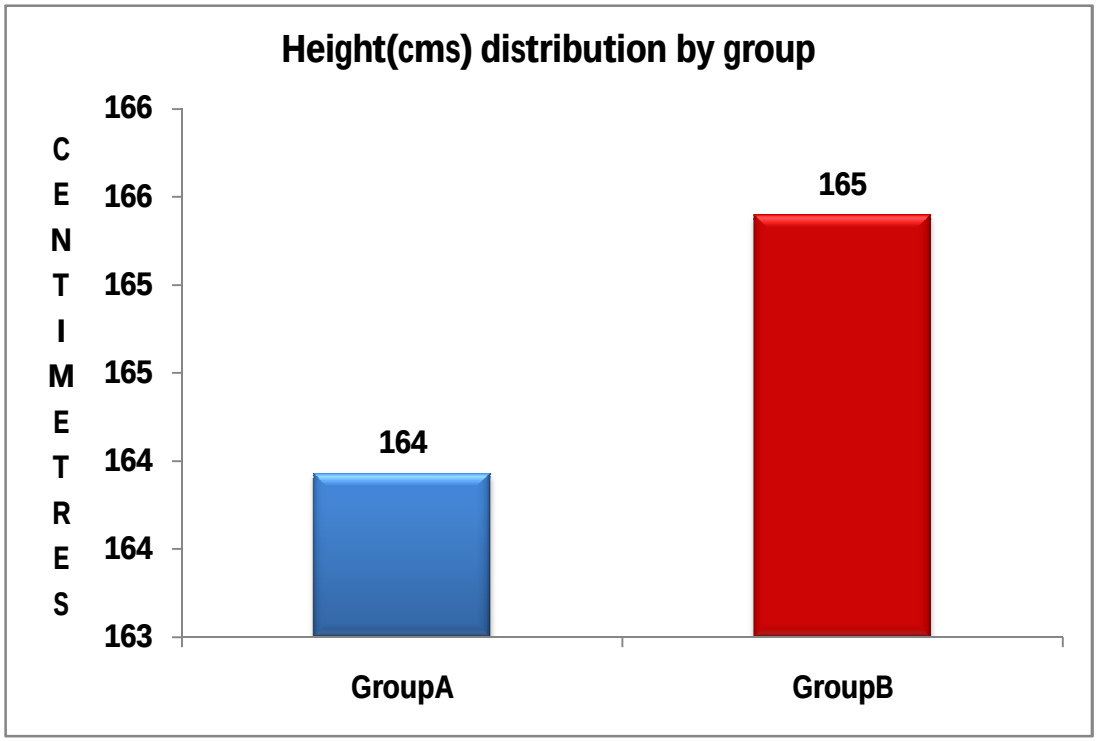
P value-1.000



HEIGHT DISTRIBUTION

The difference in height distribution among the two groups was not statistically significant, P value being 0.166.

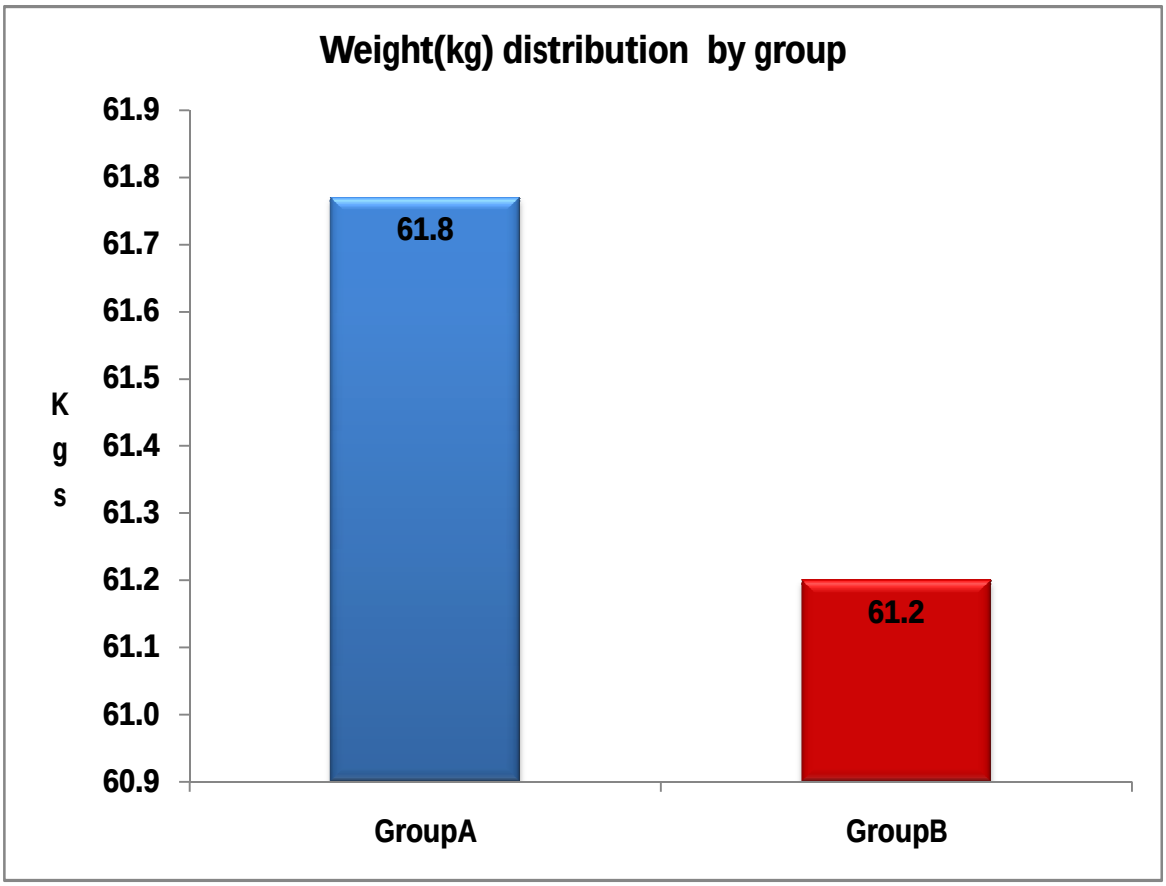
HEIGHT (IN CENTIMETRES)			
GROUP	**MEAN**	**STANDARD DEVIATION**	**p-VALUE**
A	163.93	4.193	0.166
B	165.40	3.900	



WEIGHT DISTRIBUTION

The difference in weight distribution among the two groups was not statistically significant, P value being 0.667.

WEIGHT (IN KG)			
GROUP	**MEAN**	**STANDARD DEVIATION**	**p-VALUE**
A	61.77	5.250	0.667
B	61.20	4.895	

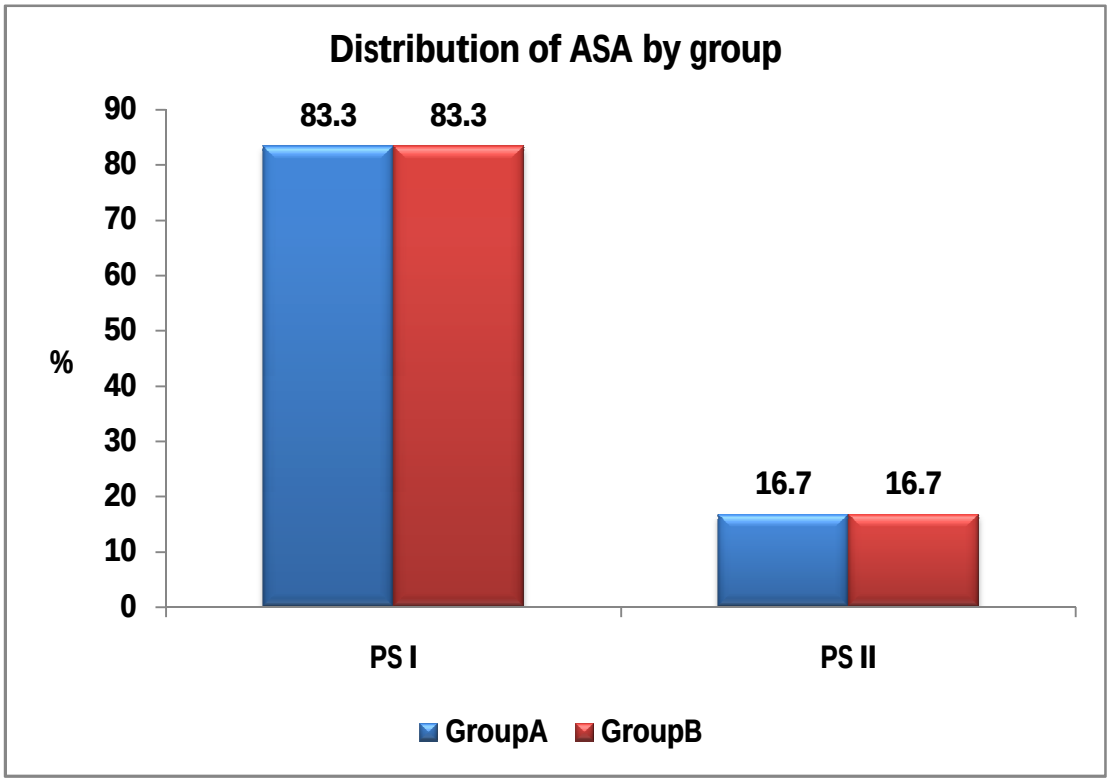


ASA STATUS

The ASA status was not statistically significant among the two groups.

	PS I		PS II	
GROUP	**Number**	**%**	**Number**	**%**
A	25	83.3	5	16.7
B	25	83.3	5	16.7

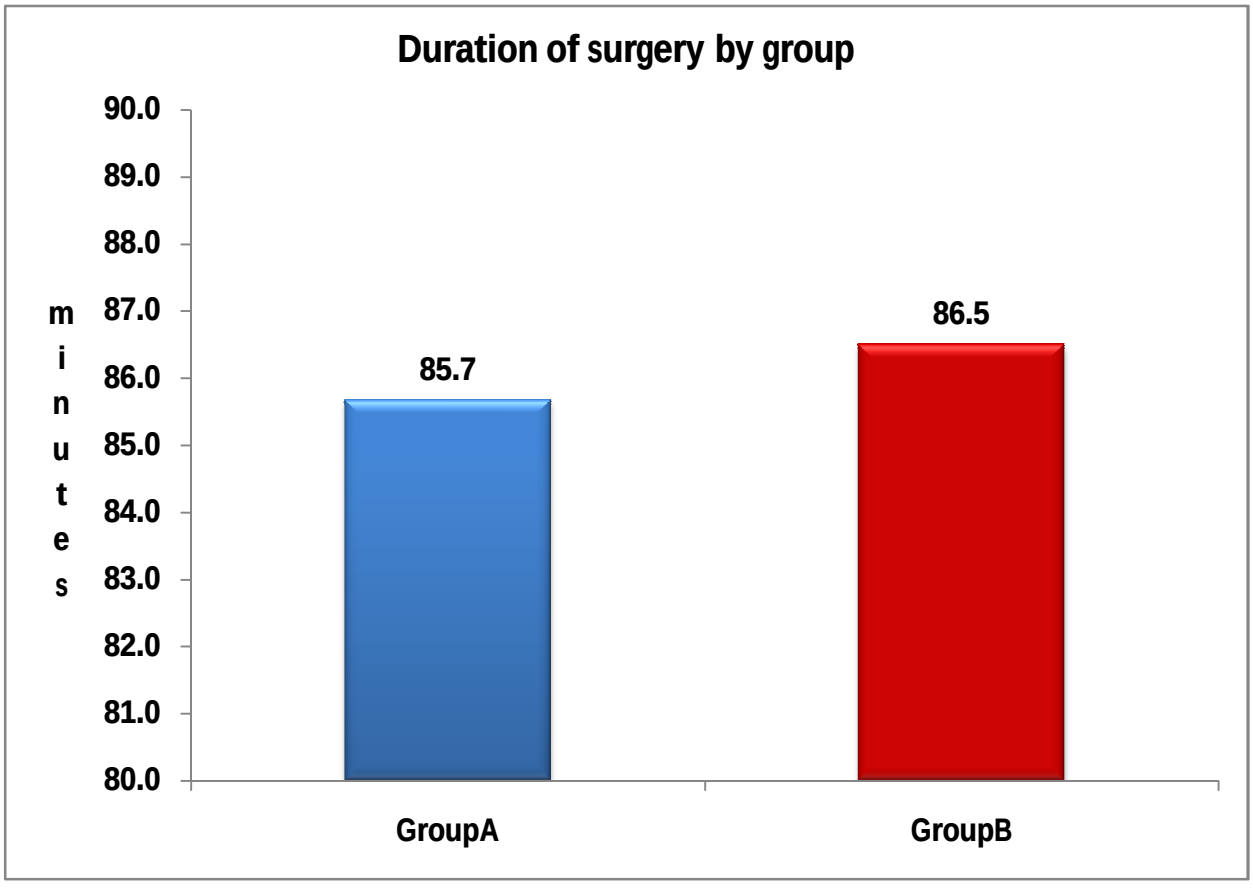
P=1.000



DURATION OF SURGERY

There was no statistically significant difference between the two groups in the duration of surgery with P value being 0.856.

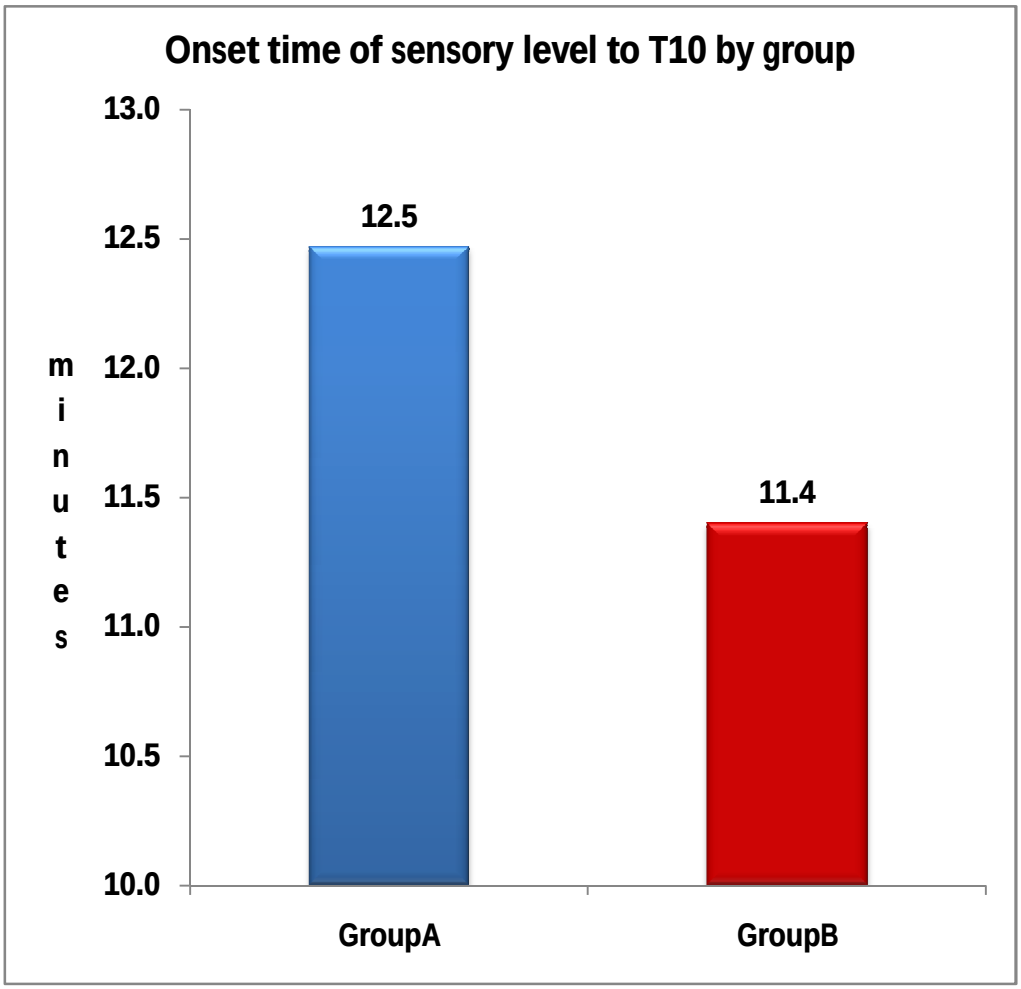
DURATION OF SURGERY (IN MINUTES)			
GROUP	**MEAN**	**STANDARD DEVIATION**	**p-VALUE**
A	85.67	15.241	0.856
B	86.50	19.963	



TIME OF ONSET OF SENSORY BLOCK TO T10

The time of onset of sensory block to T10 level was slightly lesser with group B (11.4 minutes) than group A (12.5 minutes) but was not statistically significant between the two groups with p value being 0.224.

TIME OF ONSET OF SENSORY BLOCK TO T10 LEVEL (IN MINUTES)			
GROUP	**MEAN**	**STANDARD DEVIATION**	**p-VALUE**
A	12.47	3.560	0.224
B	11.40	3.158	



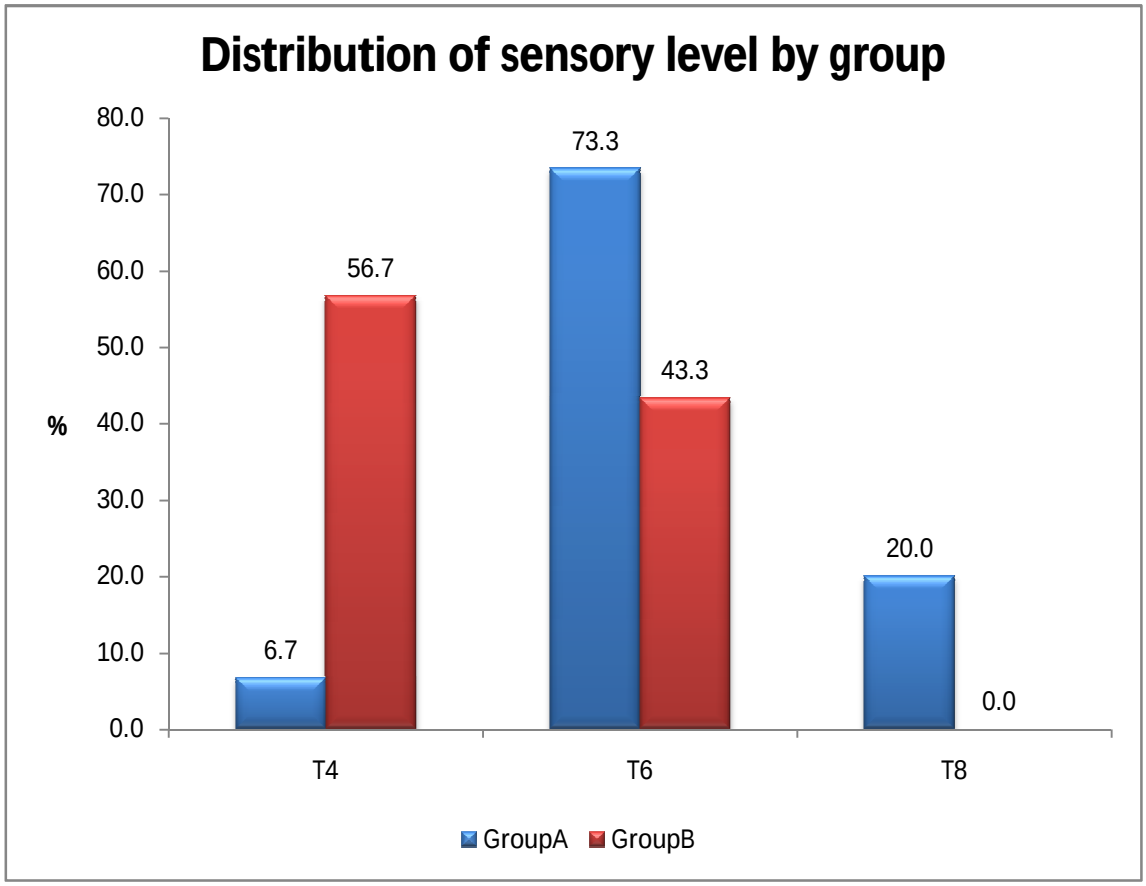
MAXIMUM SENSORY HEIGHT

The maximum sensory height attained was T4-T6 for group B and T6-T8 for group A with much statistical difference between the 2 groups **(p value-0.00004)**

DISTRIBUTION OF SENSORY BLOCK BY GROUP

	T4		T6		T8	
GROUP	**NUMBER**	**%**	**NUMBER**	**%**	**NUMBER**	**%**
A	2	6.7	22	73.3	6	20.0
B	17	56.7	13	43.3	0	0.0

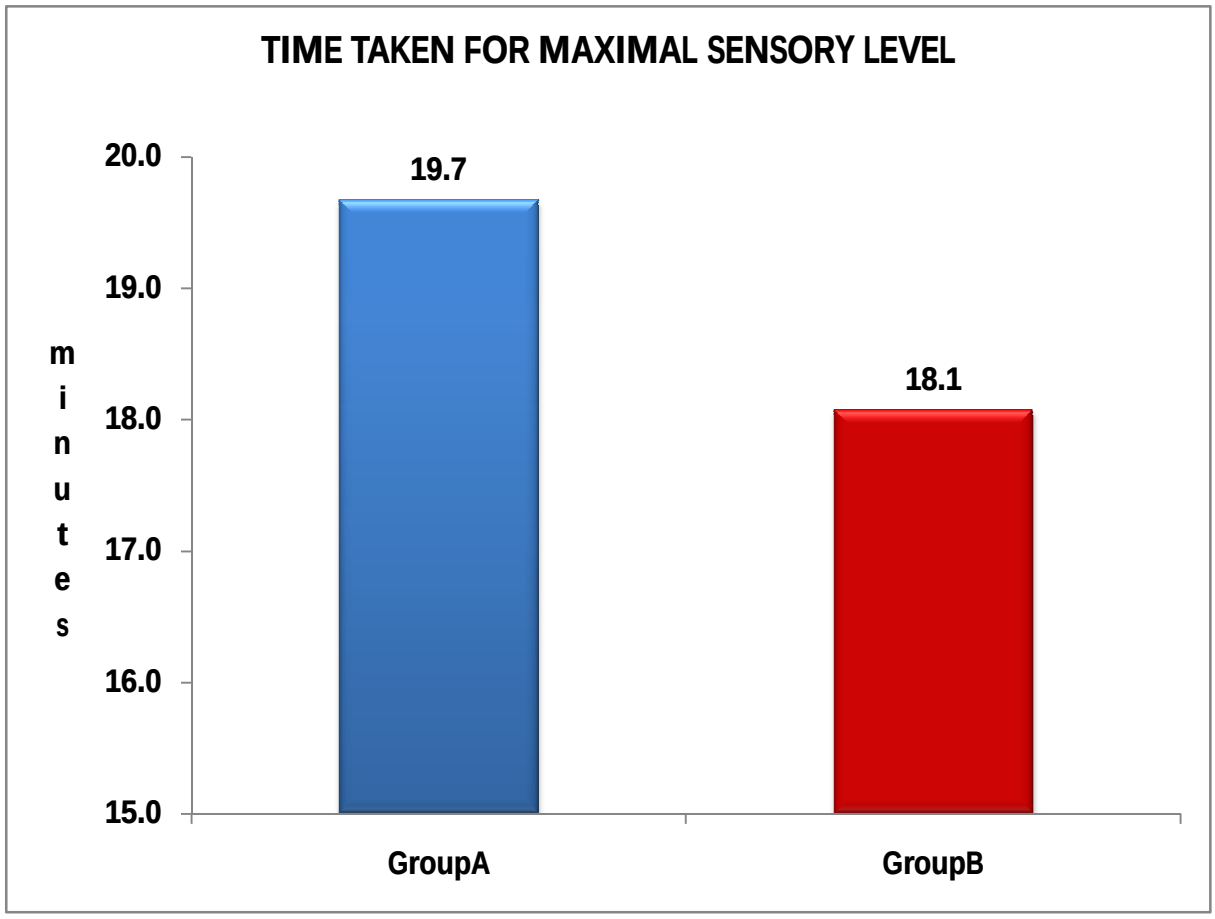
p value=0.00004



TIME TAKEN TO ACHIEVE MAXIMUM SENSORY HEIGHT

The time taken to achieve maximum sensory height was shorter with group B (18.1 minutes) when compared to group A (19.7minutes) but was statistically insignificant with p value of 0.126.

TIMETAKEN TO ACHIEVE MAXIMUM SENSORY LEVEL(IN MINUTES)			
GROUP	**MEAN**	**STANDARD DEVIATION**	**p-VALUE**
A	19.67	3.604	0.126
B	18.07	4.346	

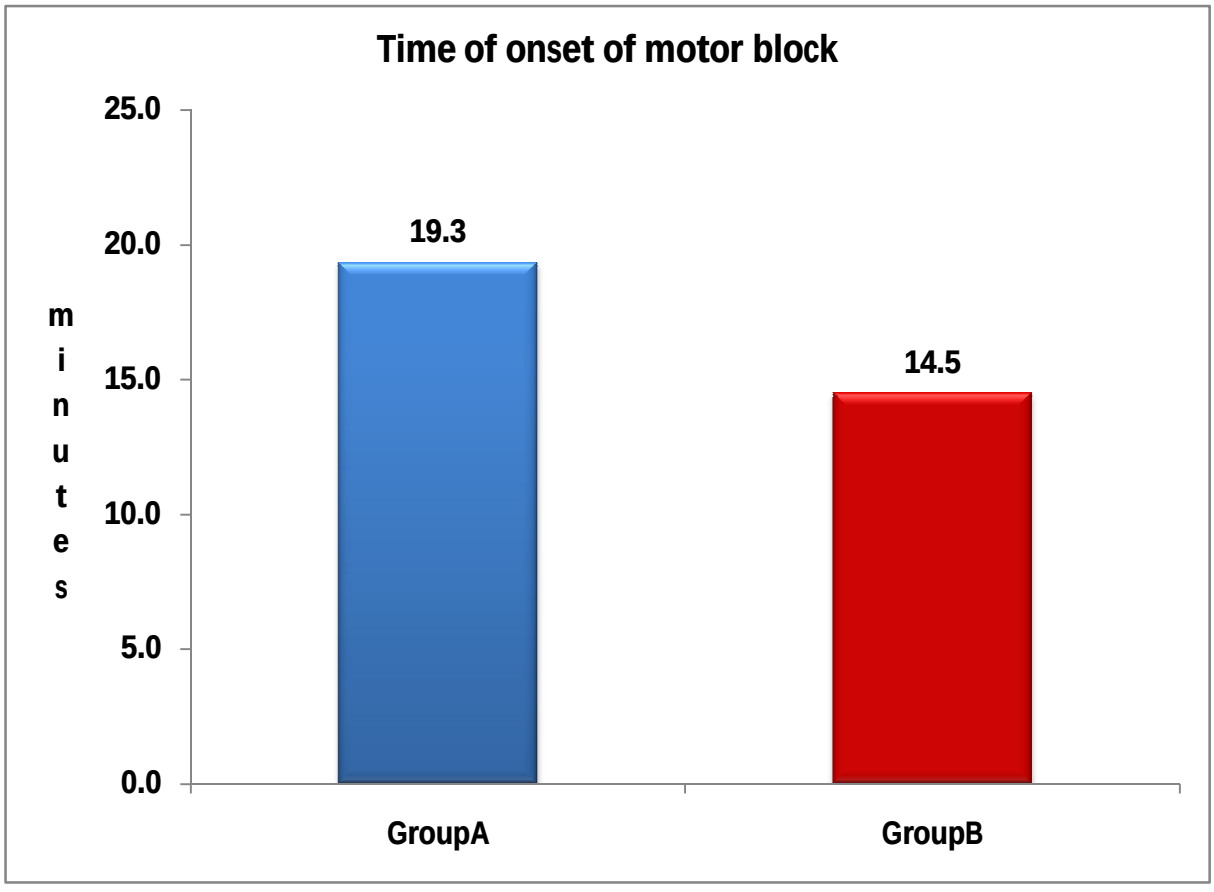


TIME OF ONSET OF MOTOR BLOCKADE

(BROMAGE SCALE 1)

The time of onset of motor blockade is the time taken to achieve Bromage scale 1 which was statistically significant between the two groups with p value of **0.000.**

ONSET TIME FOR MOTOR BLOCKADE (IN MINUTES)			
GROUP	**MEAN**	**STANDARD DEVIATION**	**p-VALUE**
A	19.33	4.498	**0.000**
B	14.50	3.560	



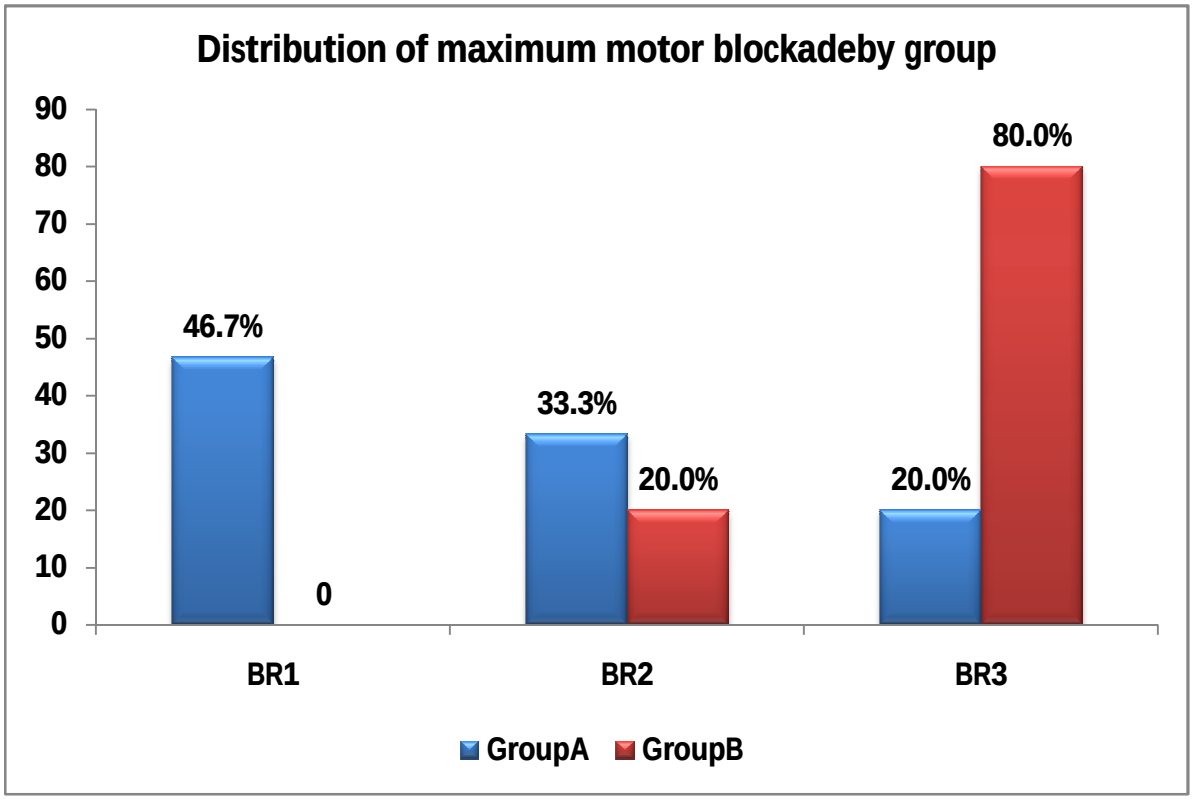
MAXIMUM MOTOR BLOCKADE ACHIEVED

Maximum motor blockade achieved showed statistical significance(p value **0.0000** between the two groups with bromage scale of 2 to 3 achieved in group B and Bromage scale of 1 to 2 achieved in group A.

DISTRIBUTION OF MOTOR BLOCK BY GROUP

	T4		T6		T8	
GROUP	**NUMBER**	**%**	**NUMBER**	**%**	**NUMBER**	**%**
A	14	46.67	10	33.3	6	20.0
B			6	20.0	24	80.0

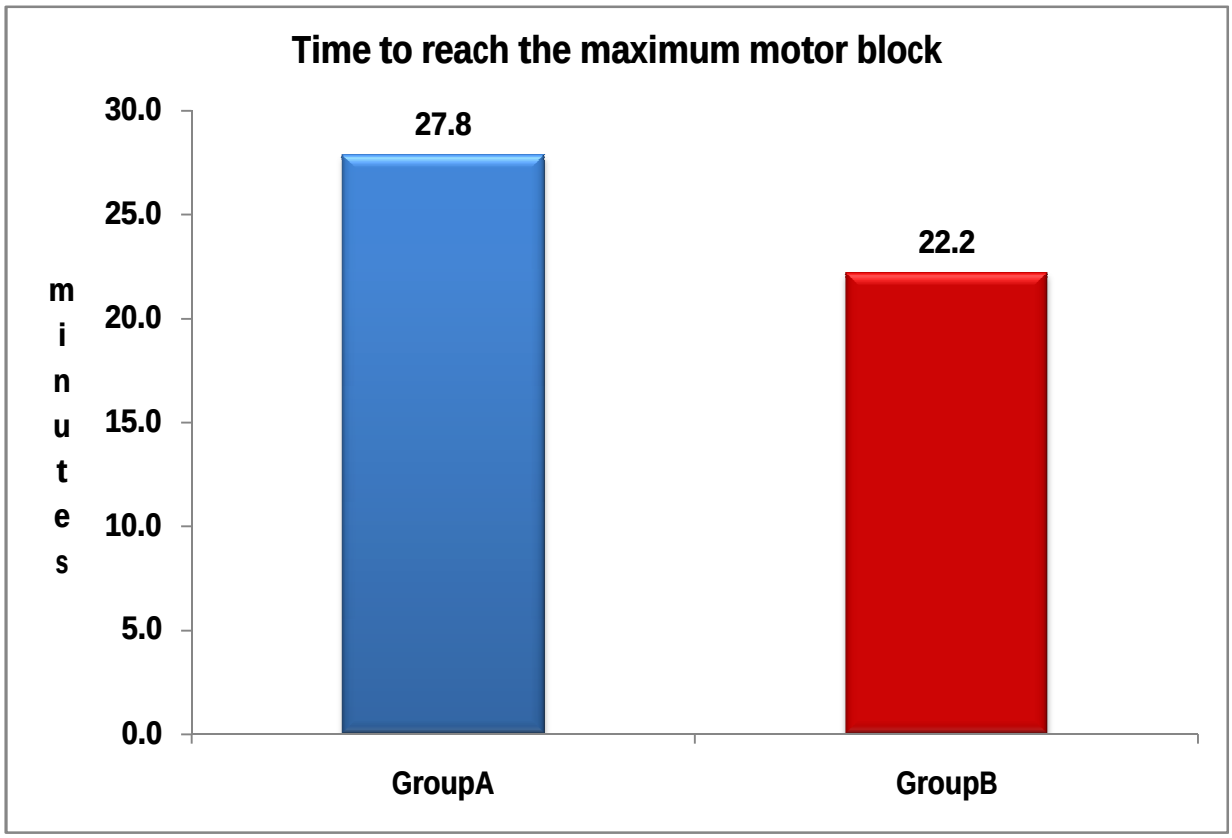
p value 0.0000



TIME TAKEN TO ACHIEVE MAXIMAL MOTOR BLOCKADE

Time taken to achieve maximum motor blockade was significantly shorter with group B (22.17 minutes) compared to group A (27.83 minutes) which is statistically significant **(p value-0.006).**

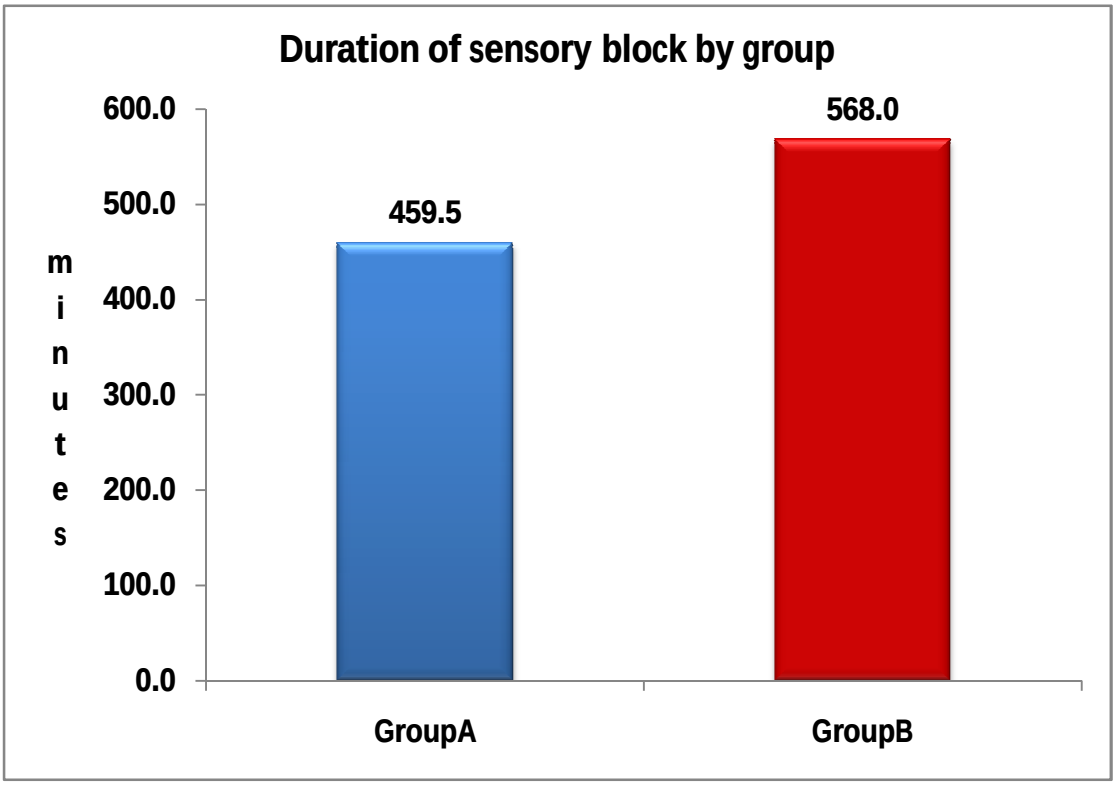
TIME TAKEN TO ACHIEVE MAXIMAL MOTOR BLOCKADE (IN MINUTES)			
GROUP	**MEAN**	**STANDARD DEVIATION**	**p-VALUE**
A	27.83	5.522	**0.006**
B	22.17	4.488	



DURATION OF SENSORY BLOCKADE

The duration of sensory blockade was defined as time taken for regression of sensory blockade to S1 level which was prolonged in group B (568 minutes) when compared to group A (459.5 minutes) and was statistically significant, p value **0.000**

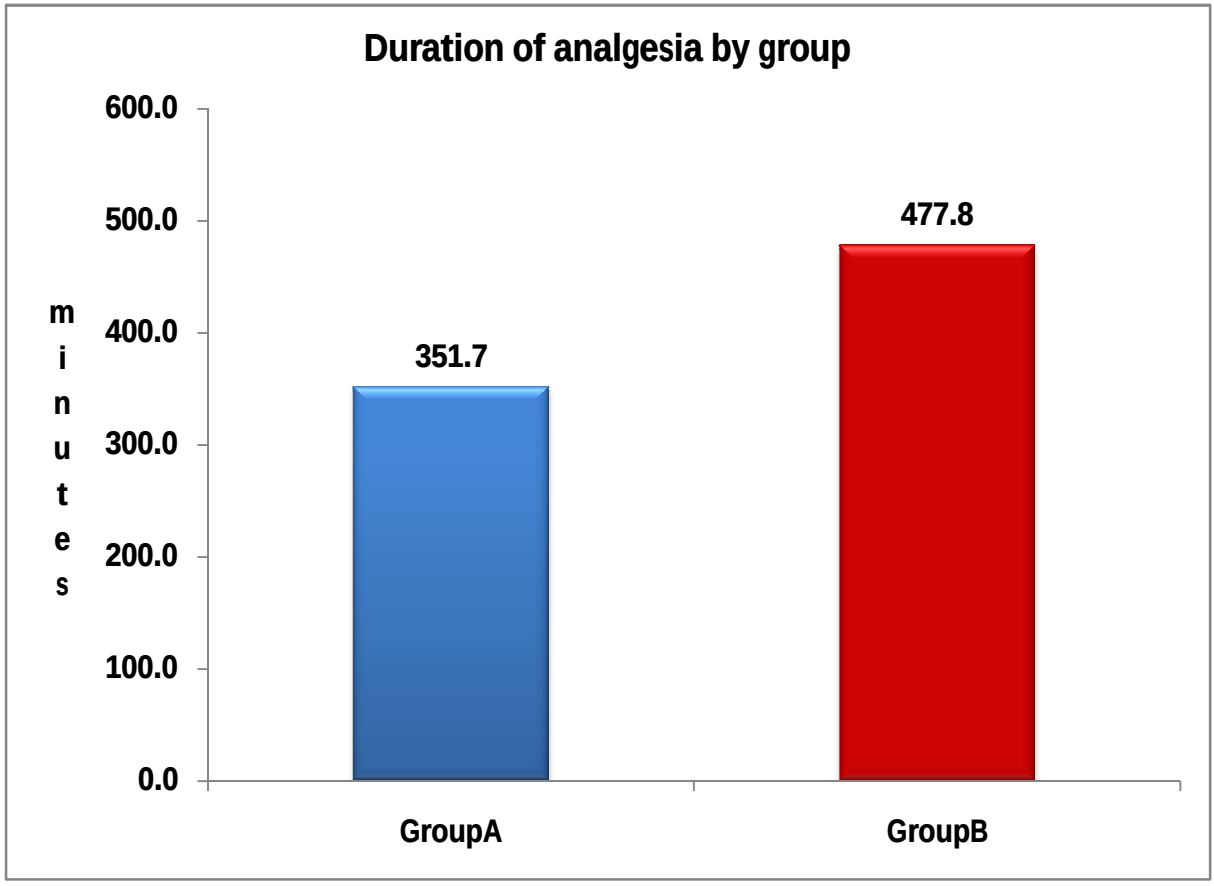
DURATION OF SENSORY BLOCKADE (IN MINUTES)			
GROUP	**MEAN**	**STANDARD DEVIATION**	**p-VALUE**
A	459.5	49.119	**0.000**
B	568	44.346	



DURATION OF ANALGESIA

Duration of analgesia is prolonged with group B (477.83 minutes) when compared to group A (351.67 minutes) which shows statistical significance with p value of **0.000.**

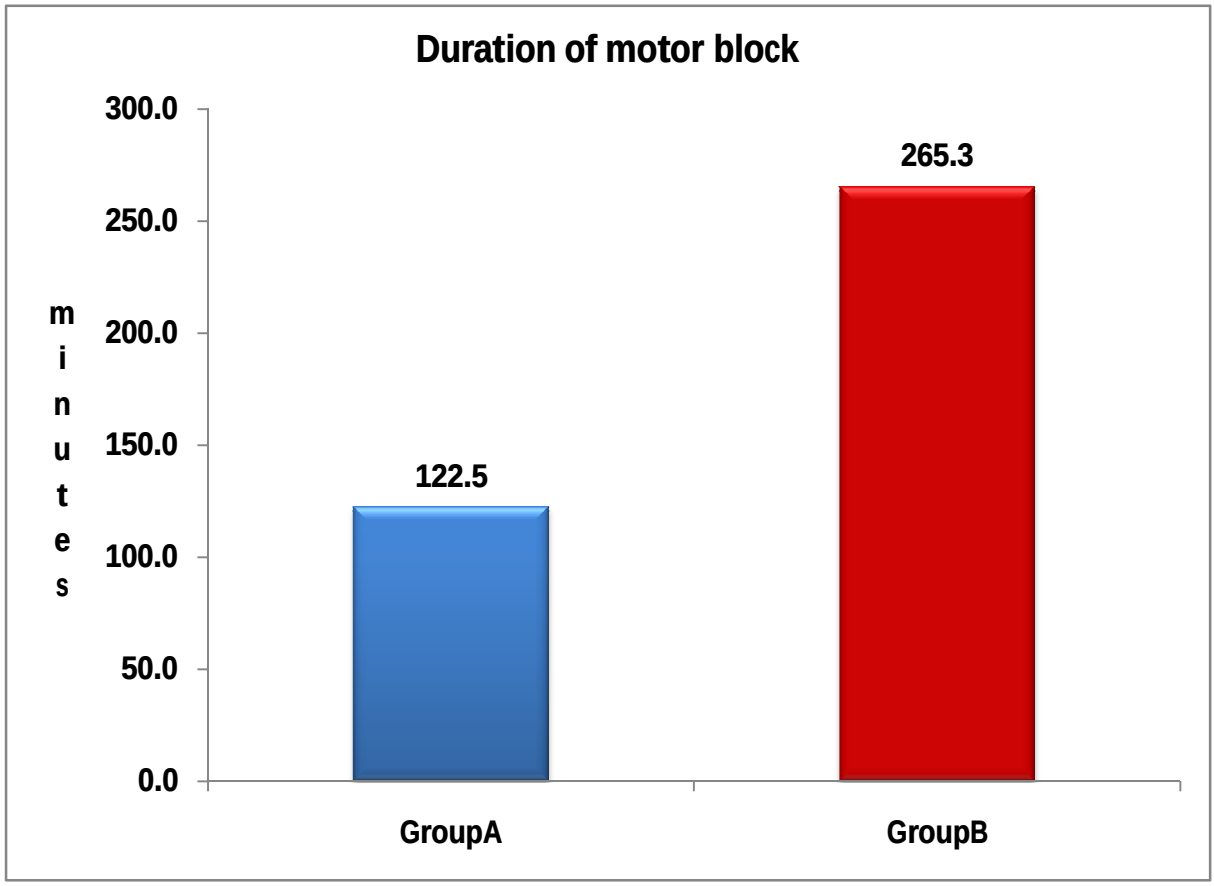
DURATION OF ANALGESIA (IN MINUTES)			
GROUP	**MEAN**	**STANDARD DEVIATION**	**p-VALUE**
A	351.67	54.730	**0.000**
B	477.83	44.580	



DURATION OF MOTOR BLOCKADE

The time duration of motor blockade is taken as the time for the motor blockade to return to Modified Bromage scale 0 and was statistically significant between two groups P value of **0.000.**

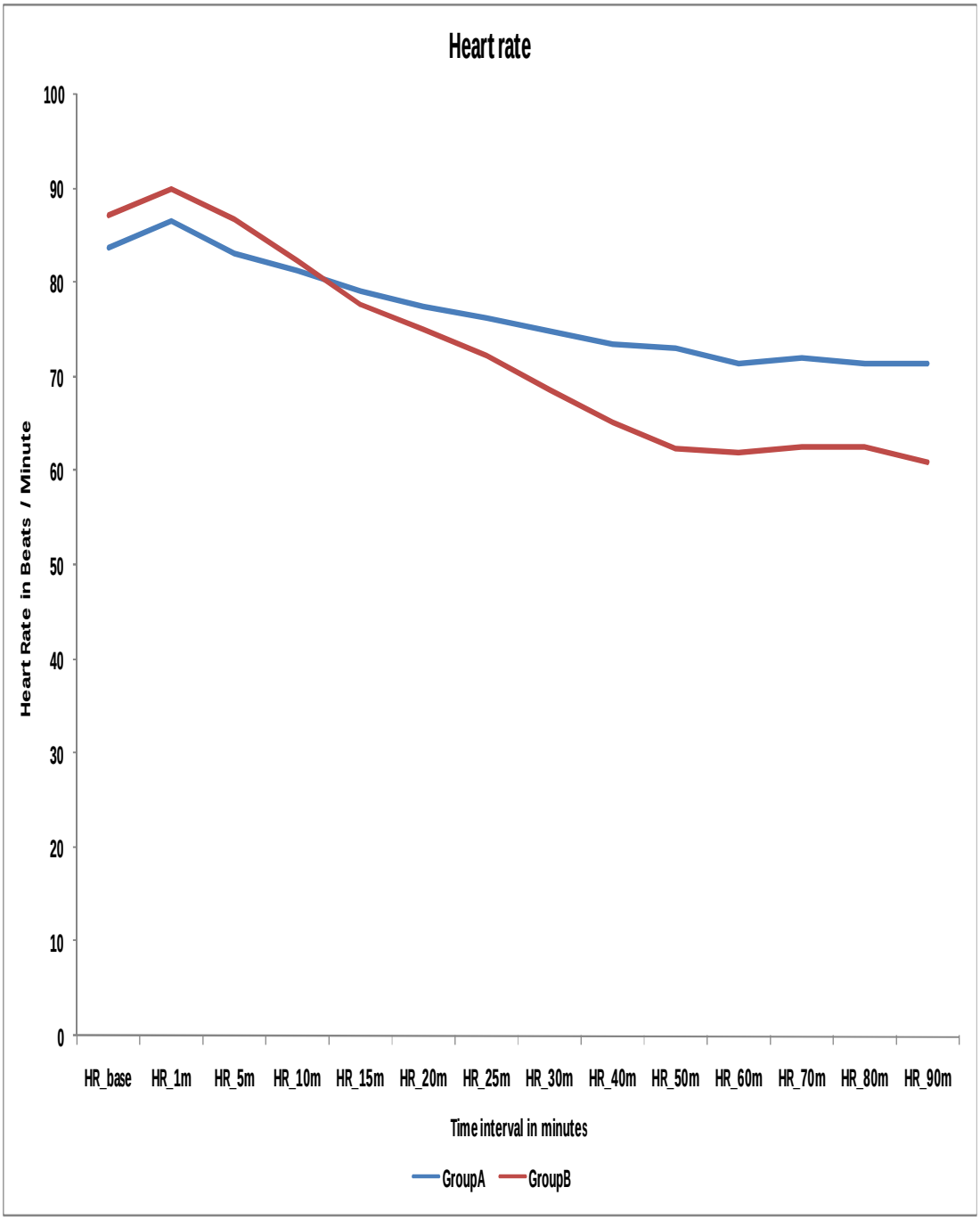
DURATION OF SENSORY BLOCKADE (IN MINUTES)			
GROUP	**MEAN**	**STANDARD DEVIATION**	**p-VALUE**
A	127.5	24.309	**0.000**
B	265.33	35.183	



HEART RATE

The heart rate recorded between 25 to 100 minutes was found to be statistically significant between the 2 groups.

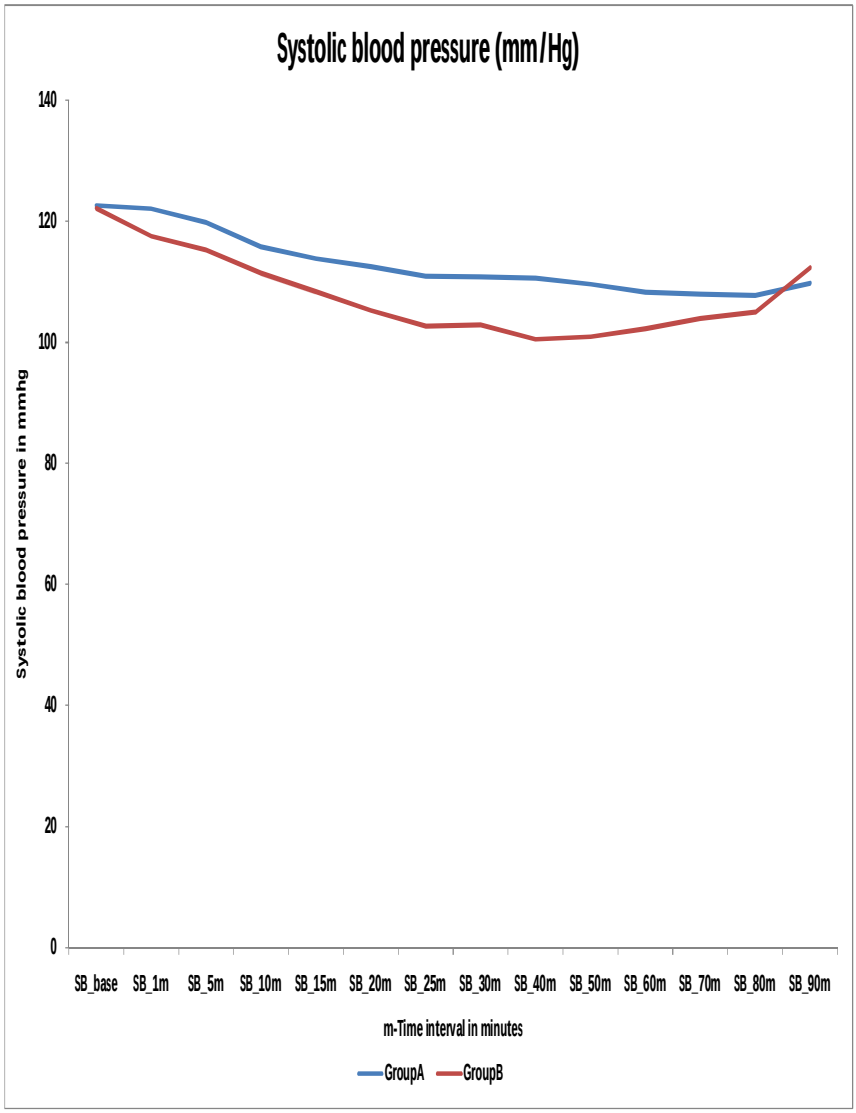
TIME INTERVAL (MINUTES)	GROUP	MEAN HEART RATE(BEATS/MINUTE)	STANDARD DEVIATION	p-VALUE
Basal	A B	83.73 87.20	6.384 7.622	0.061
1	A B	86.60 89.93	6.871 6.357	0.056
5	A B	83.10 86.73	7.355 7.763	0.068
10	A B	81.40 82.27	6.371 6.362	0.600
15	A B	79.13 77.67	6.678 7.845	0.439
20	A B	78.87 77.47	6.522 7.780	0.442
25	A B	76.20 72.20	5.616 8.006	**0.029**
30	A B	74.87 68.60	5.625 7.185	**0.000**
40	A B	73.53 65.13	6.616 7.514	**0.000**
50	A B	72.93 62.33	6.074 7.685	**0.000**
60	A B	71.47 62.07	5.823 6.918	**0.000**
70	A B	72.00 62.64	4.751 5.813	**0.000**
80	A B	71.50 62.59	4.628 6.073	**0.000**
90	A B	71.41 61.00	3.519 3.658	**0.000**
100	A B	70.25 62.00	5.175 3.225	**0.000**
110	A B	68.50 63.20	4.726 5.215	0.156
120	A B	70.00 62.67	2.000 3.055	0.025


Heart rate
100
90
80
70
60
50
40
30
20
10
0
Heart Rate in Beats / Minute
HR_base
HR_1m
HR_5m
HR_10m
HR_15m
HR_20m
HR_25m
HR_30m
HR_40m
HR_50m
HR_60m
HR_70m
HR_80m
HR_90m
Time interval in minutes
GroupA
GroupB

SYSTOLIC BLOOD PRESSURE:

The systolic blood pressures recorded at 15,20,25,30,40,50,60 minutes were significant statistically.

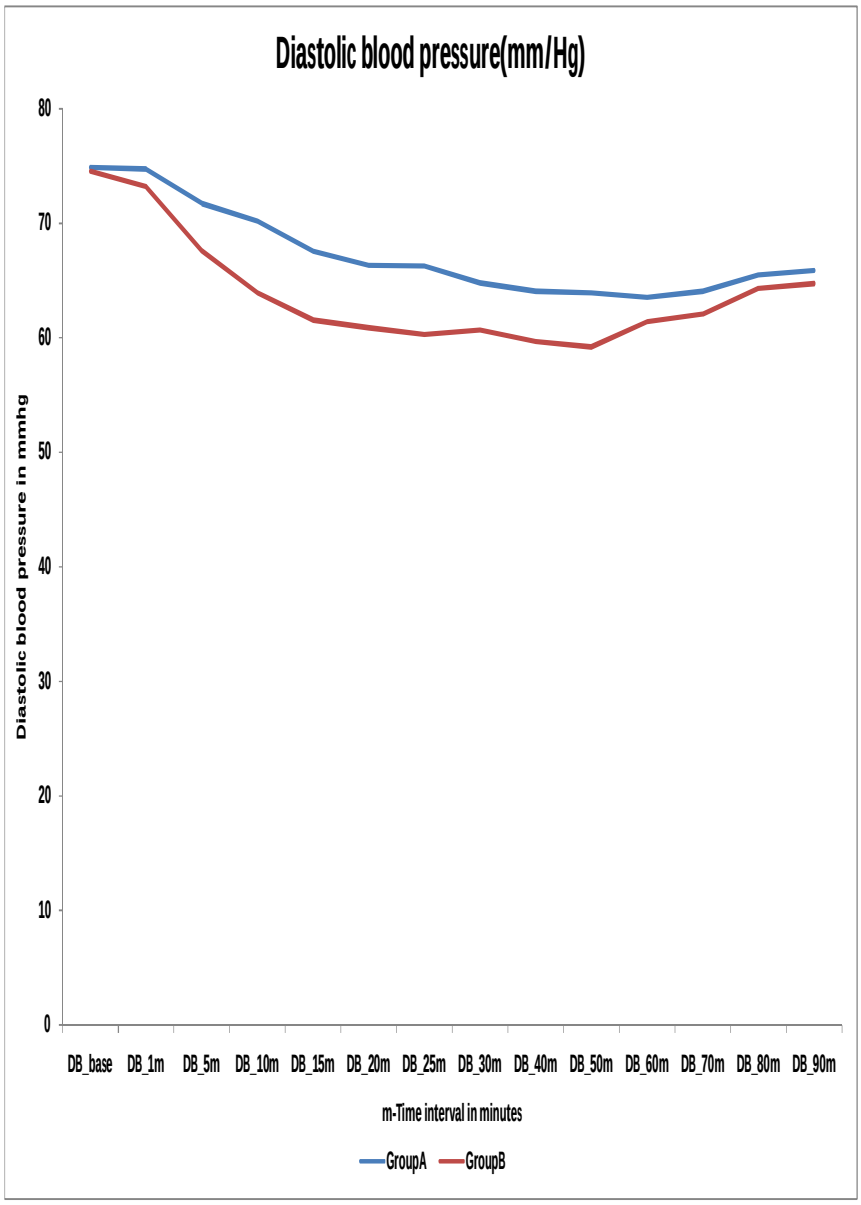
TIME INTERVAL (MINUTES)	GROUP	MEAN SYSTOLIC BLOOD PRESSURE (MM HG)	STANDARD DEVIATION	p-VALUE
Basal	A B	122.60 122.13	9.427 7.718	0.835
1	A B	122.07 117.53	8.952 22.442	0.308
5	A B	119.80 115.27	9.223 8.686	0.055
10	A B	115.80 111.47	7.034 10.621	0.068
15	A B	113.83 118.40	7.297 10.026	**0.020**
20	A B	112.53 105.27	8.337 10.272	**0.004**
25	A B	110.87 102.67	9.153 11.009	**0.003**
30	A B	110.80 102.87	9.650 11.334	**0.005**
40	A B	110.60 100.53	9.220 11.826	**0.001**
50	A B	109.60 100.93	9.676 11.026	**0.002**
60	A B	108.20 102.27	10.230 9.288	**0.022**
70	A B	107.93 104.00	7.454 10.066	0.099
80	A B	107.70 105.00	8.927 11.402	0.419
90	A B	109.76 112.36	9.641 35.763	0.776
100	A B	109.75 107.82	7.741 9.527	0.644
110	A B	109.50 106.00	7.724 10.392	0.594
120	A B	104.00 102.00	2.000 2.000	0.288


Systolic blood pressure (mm/Hg)
Systolic blood pressure in mmhg
140
120
100
80
60
40
20
0
SB_base
SB_1m
SB_5m
SB_10m
SB_15m
SB_20m
SB_25m
SB_30m
SB_40m
SB_50m
SB_60m
SB_70m
SB_80m
SB_90m
m-Time interval in minutes
GroupA
GroupB

DIASTOLIC BLOOD PRESSURE

In this study the diastolic blood pressure recorded at 5, 10,15,20,25,30,40,50 minutes were statistically significant.

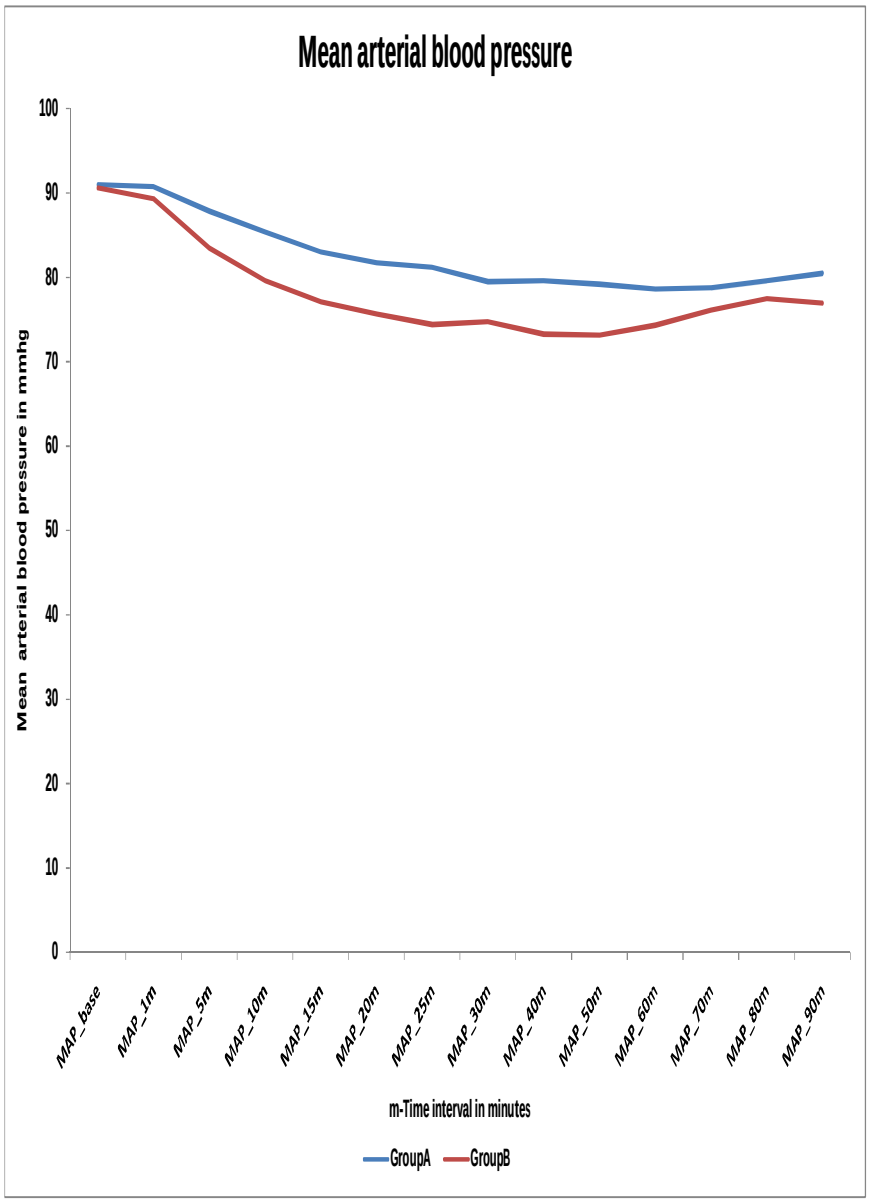
TIME INTERVAL (MINUTES)	GROUP	MEAN DIASTOLIC BLOOD PRESSURE (MM HG)	STANDARD DEVIATION	p-VALUE
Basal	A B	74.87 74.53	6.027 6.345	0.835
1	A B	74.73 73.20	6.443 7.327	0.393
5	A B	71.73 67.60	5.747 7.190	**0.017**
10	A B	70.20 63.93	5.762 7.511	**0.001**
15	A B	67.53 61.53	5.698 6.637	**0.000**
20	A B	66.33 60.87	6.870 7.855	**0.006**
25	A B	66.27 60.27	6.963 6.680	**0.001**
30	A B	64.80 60.67	7.730 7.919	**0.045**
40	A B	64.07 59.67	7.153 9.686	**0.050**
50	A B	63.93 59.20	7.904 7.959	**0.024**
60	A B	63.53 61.40	8.148 7.486	0.295
70	A B	64.07 62.07	7.662 6.049	0.280
80	A B	65.50 64.33	7.619 5.099	0.587
90	A B	65.88 64.71	7.193 6.866	0.650
100	A B	67.50 65.82	6.655 5.964	0.571
110	A B	66.00 67.20	6.733 9.654	0.840
120	A B	64.00 63.33	4.000 5.033	0.866


Diastolic blood pressure(mm/Hg)
Diastolic blood pressure in mmhg
80
70
60
50
40
30
20
10
0
DB_base
DB_1m
DB_5m
DB_10m
DB_15m
DB_20m
DB_25m
DB_30m
DB_40m
DB_50m
DB_60m
DB_70m
DB_80m
DB_90m
m-Time interval in minutes
GroupA
GroupB

MEAN ARTERIAL BLOOD PRESSURE

Mean arterial blood pressure recorded at 5, 10, 15,20,25,30,40,50,60 minutes were found to be statistically significant.

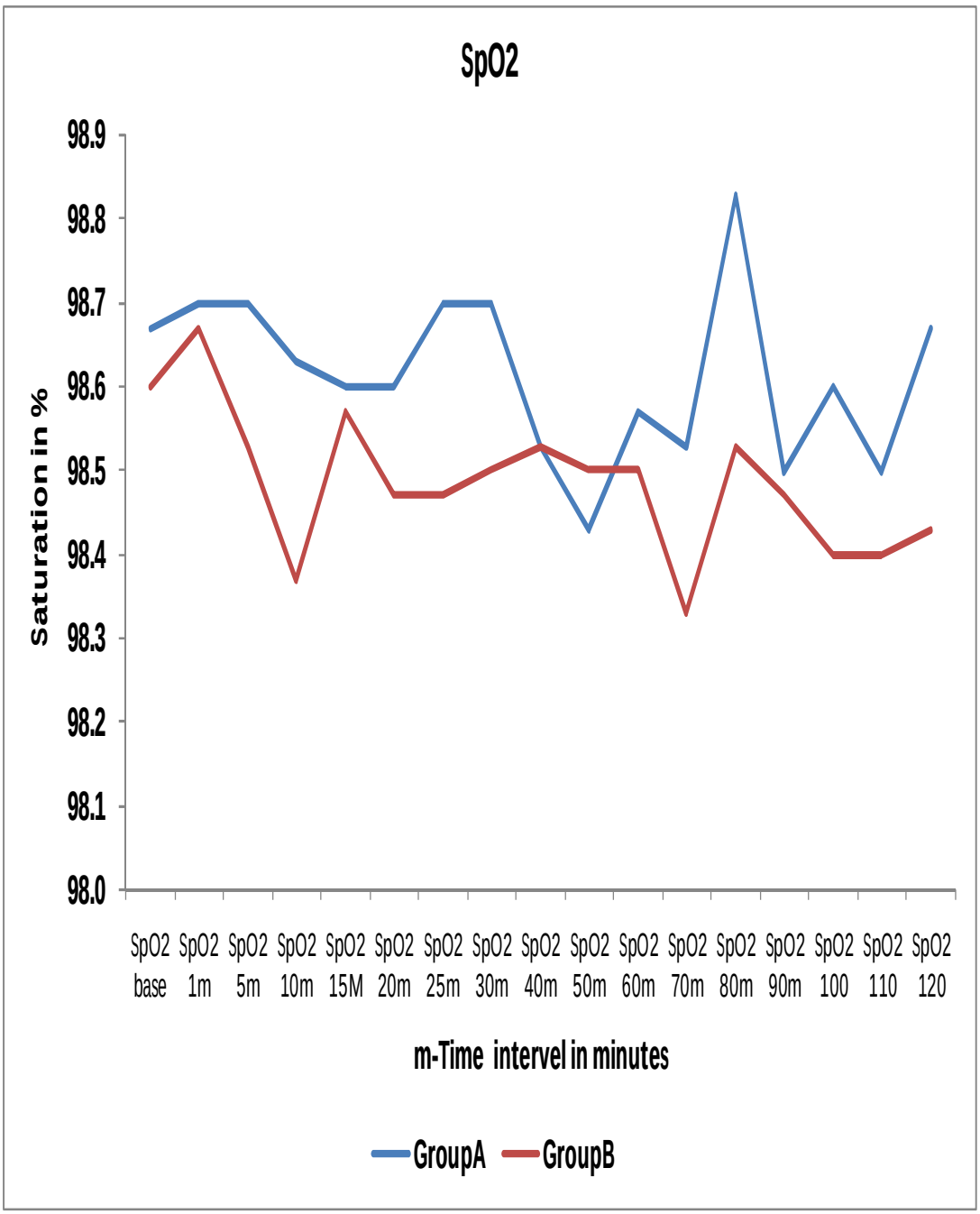
TIME INTERVAL (MINUTES)	GROUP	MEAN OF MEAN ARTERIAL BLOOD PRESSURE (MM HG)	STANDARD DEVIATION	p-VALUE
Basal	A B	90.97 90.60	5.327 5.143	0.787
1	A B	90.73 89.27	5.105 6.085	0.316
5	A B	87.83 83.47	5.497 6.816	**0.008**
10	A B	85.33 79.60	4.751 8.067	**0.002**
15	A B	83.03 77.10	4.664 6.989	**0.000**
20	A B	81.73 75.67	5.977 8.049	**0.002**
25	A B	81.20 74.40	6.718 7.403	**0.000**
30	A B	79.50 74.70	7.385 8.014	**0.019**
40	A B	79.57 73.27	6.831 9.791	**0.005**
50	A B	79.17 73.13	7.525 8.402	**0.005**
60	A B	78.63 74.30	7.872 7.603	**0.034**
70	A B	78.76 76.14	6.162 6.604	0.128
80	A B	79.65 77.47	7.307 7.985	0.392
90	A B	80.47 76.93	7.383 8.204	0.216
100	A B	81.62 78.55	5.829 8.745	0.400
110	A B	80.50 80.00	6.856 9.798	0.934
120	A B	77.67 76.33	3.055 4.041	0.672


Mean arterial blood pressure
Mean arterial blood pressure in mmhg
100
90
80
70
60
50
40
30
20
10
0
MAP_base
MAP_1m
MAP_5m
MAP_10m
MAP_15m
MAP_20m
MAP_25m
MAP_30m
MAP_40m
MAP_50m
MAP_60m
MAP_70m
MAP_80m
MAP_90m
m-Time interval in minutes
GroupA
GroupB

OXYGEN SATURATION: SpO_2

There was no significant difference between the two groups with regards to oxygen saturation.

TIME INTERVAL (MINUTES)	GROUP	MEAN OF SpO_2 (%)	STANDARD DEVIATION	p-VALUE
Basal	A	98.67	0.661	0.676
	B	98.60	0.563	
1	A	98.70	0.651	0.831
	B	98.67	0.547	
5	A	98.70	0.535	0.221
	B	98.53	0.507	
10	A	98.63	0.556	0.054
	B	98.37	0.490	
15	A	98.60	0.675	0.829
	B	98.57	0.504	
20	A	98.60	0.563	0.339
	B	98.47	0.507	
25	A	98.70	0.596	0.108
	B	98.47	0.507	
30	A	98.70	0.651	0.190
	B	98.50	0.509	
40	A	98.53	0.571	1.000
	B	98.53	0.507	
50	A	98.43	0.504	0.634
	B	98.50	0.572	
60	A	98.57	0.626	0.652
	B	98.50	0.509	
70	A	98.53	0.507	0.122
	B	98.33	0.479	
80	A	98.83	0.648	0.062
	B	98.53	0.571	
90	A	98.50	0.572	0.812
	B	98.47	0.507	
100	A	98.60	0.621	0.174
	B	98.40	0.498	
110	A	98.50	0.572	0.473
	B	98.40	0.498	
120	A	98.67	0.606	0.111
	B	98.43	0.504	


SpO2
98.9
98.8
98.7
98.6
98.5
98.4
98.3
98.2
98.1
98.0
Saturation in %
SpO2 base
SpO2 1m
SpO2 5m
SpO2 10m
SpO2 15M
SpO2 20m
SpO2 25m
SpO2 30m
SpO2 40m
SpO2 50m
SpO2 60m
SpO2 70m
SpO2 80m
SpO2 90m
SpO2 100
SpO2 110
SpO2 120
m-Time intervel in minutes
GroupA
GroupB

ADVERSE EFFECTS

Adverse effects were reported in 16.7% of patients in group A and 30% of patients in group B.But hypotension/bradycardia (fall of >20% from the baseline) requiring treatment with Inj Ephedrine / Inj Atropine) were found to be statistically insignificant while comparing the two groups.

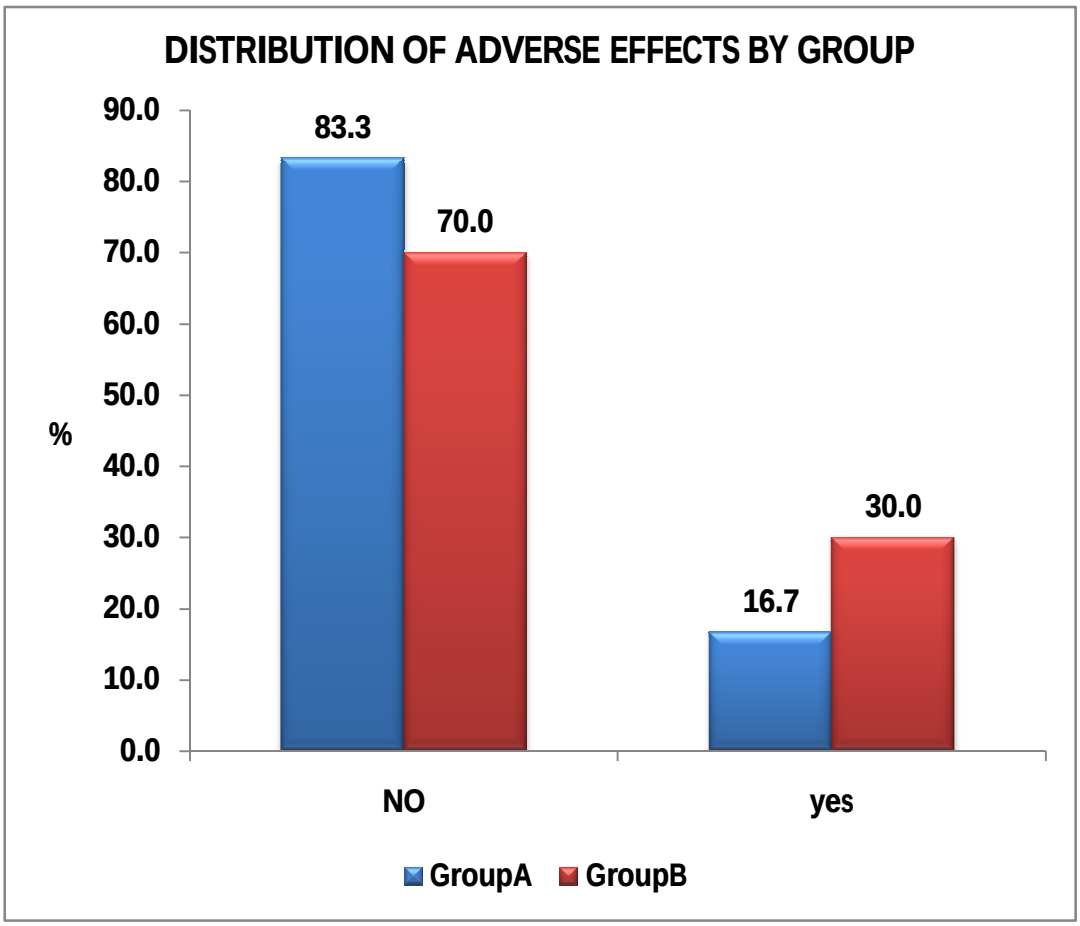
DISTRIBUTION OF ADVERSE EFFECTS IN BOTH GROUPS

ADVERSE EFFECTS		GROUP	
		Group A	Group B
Dry Mouth	Count % within Group	-	1 3.33%
Nausea Vomiting	Count % within Group	1 3.33%	1 3.33%
Shivering	Count % within Group	-	2 6.67%
Urinary Retention	Count % within Group	2 6.67%	-
Hypotension	Count % within Group	2 6.67%	3 10%
Bradycardia	Count % within Group	-	2 6.67%

OVERALL DISTRIBUTION OF ADVERSE EFFECTS

Group	Yes	%	No	%
A	5	16.67	25	83.3
B	9	30	21	70.0
Total	14	23.33	46	75.40

p-value-0.3598



ADVERSE EFFECTS REQUIRING TREATMENT IN BOTH GROUPS

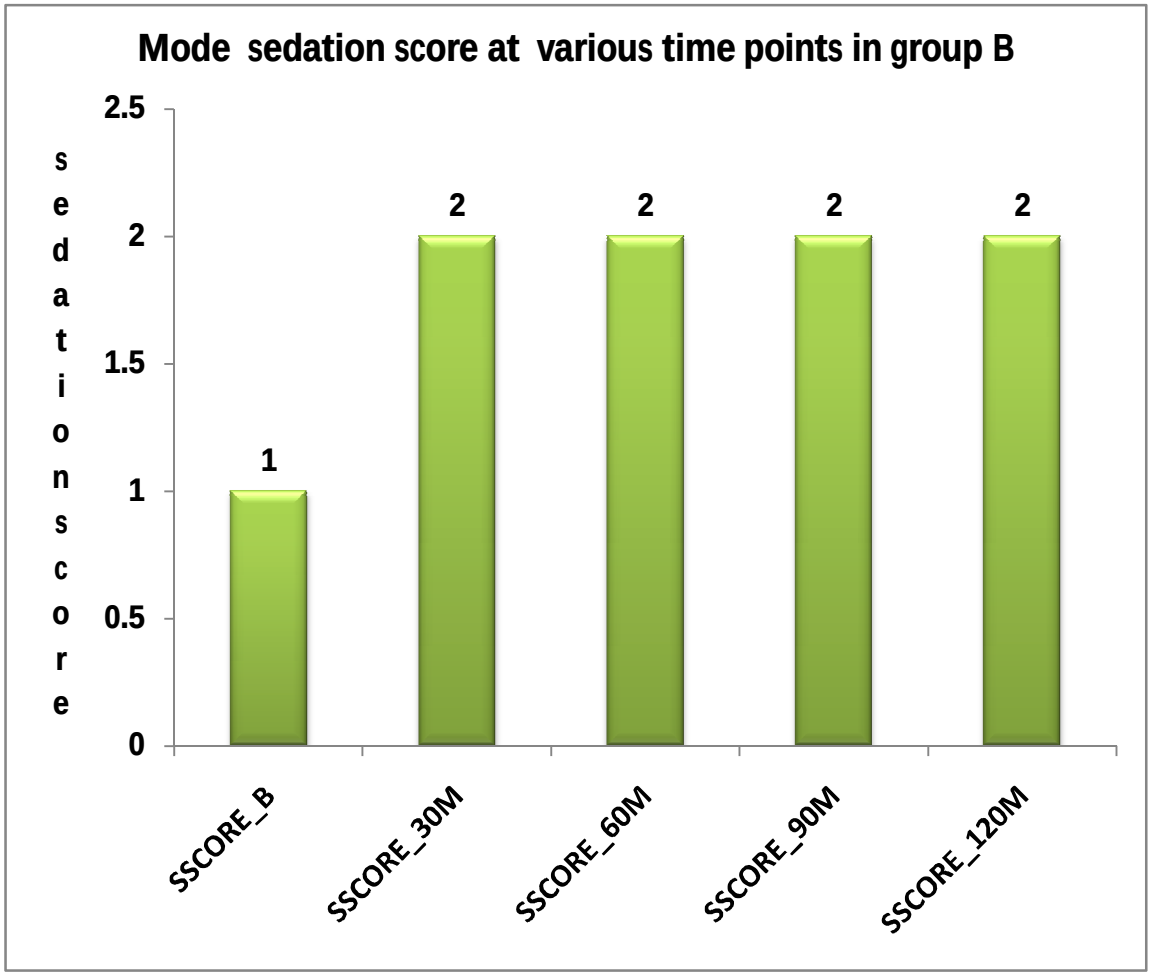
GROUP	NUMBER	%
A	2	6.67
B	5	16.67

p VALUE-0.999

SEDATION SCORE:

Mode sedation score in group B (Dexmedetomidine group) was found to be 2 at all points of time as per Ramsay scoring.

SEDATION SCORE AT VARIOUS TIME INTERVALS			
TIME INTERVAL IN MINUTES	**MEAN**	**MEDIAN**	**MODE**
BASAL	1	1	1
30	1.87	2	2
60	2.27	2	2
90	2.03	2	2
120	1.63	2	2



DISCUSSION

This study compared the effects of addition of epidural dexmedetomidine(50 micrograms) to epidural 0.5% levobupivacaine 20 ml for infraumbilical & lower limb surgeries.

1.DEMOGRAPHIC DATA

Demographic parameters such as age, sex, height & weight have no statistically significant difference between the two groups. Distribution of ASA status was also similar in both groups.

2.TIME OF ONSET OF SENSORY BLOCKADE

Onset time to sensory block at T 10 level was defined as time taken from the injection of local anaesthetic till the loss of sensation for pin prick at T10 level.In this study,mean time of onset of sensory blockade to T10 level was 12.47 minutes for group A & 11.4 minutes for group B which shows that the onset is slightly earlier with group B but was not found to be statistically significant.(p value 0.224)

Kopacz et al(2001)[46] in their study found the mean time of onset of sensory block to T10 level to be 13 minutes with epidural 0.5% levobupivacaine 20 ml and with the addition of dexmedetomidine the mean onset time was 11.5 minutes.

3. MAXIMUM SENSORY LEVEL REACHED AND TIME TAKEN

Maximium sensory height reached ranged from T6 to T8 in group A compared to T4 to T6 in group B with p value of 0.00004 which is statistically significant.This is in accordance with the studies conducted by Cox et al[35] in which the maximum sensory levels attained were T8

and T6 with epidural 0.5% levobupivacaine and with the addition of epidural dexmedetomidine respectively. Bajwa et al[47] also found the maximum sensory level to range between T4 to T6 with the addition of epidural dexmedetomidine.

With regard to the mean time taken to achieve the maximum sensory level,it was found to be 19.67 minutes in group A and 18.07 minutes in group B which was not found to be statistically significant(p value 0.126).This correlates with the study conducted by Saurav et al[48] who showed that the time taken to achieve the uppermost sensory level with 0.5% levobupivacaine was 13.5+/-7.5 minutes and 13.38+/-4.48 minutes with the addition of epidural dexmedetomidine 50 micrograms.

4. TIME OF ONSET OF MOTOR BLOCKADE

Onset time to motor blockade has been defined as time taken to achieve motor blockade of Bromage scale 1from the time of injection of local anaesthetic. This study found the mean time to onset of motor blockade to be 19.33 minutes in group A as opposed to 14.5 minutes in group B which was statistically significant with p value of 0.000.

As already pointed out by Kopacz et al[46] and Murdoch et al[39] the onset time to motor blockade is slower with 0.5% levobupivacaine with mean onset time being 20+/-5 minutes but is hastened with the addition of dexmedetomidine(14.24+/5.52 minutes). Bajwa et al[47] also found that the onset time to motor blockade is significantly shorter with the addition of dexmedetomidine with mean time ranging between 15.14+/-4.52 minutes.

5. DEGREE OF MOTOR BLOCKADE AND TIME TAKEN TO ACHIEVE

The comparison of degree of motor blockade between the 2 groups showed statistical significance with group B patients having maximum degree of motor blockade (Bromage 3) than in group A patients where the maximum motor blockade achieved was Bromage 2.This is in concordance with the study conducted by Saurav et al[48] who concluded that the maximum scale of motor block with 0.5% levobupivacaine alone ranged between Bromage 1 to 2 as compared to Bromage scale between 2-3 achieved with the addition of dexmedetomidine.

The time taken to achieve the maximum scale of motor block also showed statistical significance between the 2 groups in this study with mean time of 27.83 minutes in group A and 22.17 minutes in group B. This has been found to correlate with the study of Bajwa et al[47] who pointed out that the time taken to achieve the maximum motor blockade was shorter with the addition of 50 micrograms of dexmedetomidine(17.24+/-5.26 minutes) rather than with levobupivacaine alone.

6. DURATION OF ANALGESIA

The mean duration of analgesia was found to be highly significant in group B(mean time- 477.83 minutes) in comparison to group A(mean time-351.67 minutes) with p value of 0.000.

7. DURATION OF SENSORY AND MOTOR BLOCKADE

Statistical significance has also been found with respect to the duration of sensory and motor blockade in the 2 groups, with the additive group showing prolonged duration of both sensory and motor blockade.

Mean durations of sensory blockade(i.e) mean time to complete regression of sensory blockade to S1 were 459.5 minutes & 568 minutes in groups A & B respectively. Mean durations of motor blockade(i.e) time taken for complete regression of motor blockade to modified Bromage scale 0 were found to be 122.5 minutes and 265.33 minutes respectively in groups A & B.

Cox et al[35] and Mantouvalou et al[49] also reported the mean duration of sensory blockade to be 460 minutes and the mean duration of motor blockade to be 122 minutes with 20 ml of 0.5% levobupivacaine used for lower limb surgeries. This mean duration of both sensory & motor blockade increased with the addition of dexmedetomidine as studied by Kamal & Talaat et al[45].

8. HEMODYNAMIC PARAMETERS

HEART RATE, SYSTOLIC,DIASTOLIC & MEAN ARTERIAL BLOOD PRESSURE

The fall in heart rate in group B was maximum between 25 minutes to 100 minutes which showed statistical significance compared to group A. But decrease in heart rate more than 20% from the baseline or less than 50 beats/min which needed treatment with Inj Atropine was found in 2 out of 30 patients in group B and was not found in any of the group A patients which is statistically insignificant.

Similarly fall in systolic, diastolic & mean arterial blood pressure in group B was maximum from 5 to 60 minutes with statistical significance compared to group A. But the fall in systolic blood pressure >20% from baseline or less than 90 mmHg which necessitated treatment with Inj Ephedrine was found in 3 patients in group B patients as compared to 2 patients in group A which is also statistically not significant. Though there was fall in systolic blood pressure <90 mmhg in 3 out of 30 patients in group B patients, their mean arterial blood pressures never went below 60 mmHg which correlates with the study conducted by Bajwa et al[47] with the use of epidural dexmedetomidine.

Also postoperatively, heart rate & blood pressure remained stable in both the groups.The stable hemodynamics can be explained on the basis of the concentration of levobupivacaine used and the selection of suitable dose of dexmedetomidine.

9. SEDATION SCORES

Epidural administration of dexmedetomidine is associated with sedation, analgesia, anxiolysis, hypnosis & sympatholysis. Mean sedation score in group B was predominantly found to be 2 as per Ramsay sedation scoring (i.e) arousable to verbal commands, at all point of times observed. Absence of respiratory depression is a remarkable feature of dexmedetomidine as pointed out by Kamal et al[45].

10. ADVERSE EFFECTS

Adverse effects were noted only in 5 out of 30 patients in group A and in 9 patients in group B which is statistically non significant.

Among group A patients, nausea, vomiting was reported in 1 patient and urinary retention was noted in 2 patients. Among group B patients, dry mouth, nausea & vomiting were observed one in each of the patients, shivering in 2 patients. Overall 30% of the patients in group B experienced adverse effects (including hypotension & bradycardia) which is statistically insignificant as compared to 16.67% in group A patients. These findings correlate well with the study conducted by Bajwa SJS, Bajwa SK et al[50] where similar adverse effects experienced in both groups were reported to be only mildly discomforting to the patients. None of the patients in group B had profound deep sedation or respiratory depression with the addition of dexmedetomidine.

SUMMARY

The present study compared the effects of addition of epidural dexmedetomidine 50 micrograms to epidural 0.5% levobupivacaine for infraumbilical and lower limb surgeries.

Sixty patients of either sex belonging to ASA I & II in the age group of 25-45 years scheduled for infraumbilical and lower limb surgeries were randomly selected and divided into 2 groups A and B.(30 each)

Group A-30 patients were randomly assigned to receive 0.5% isobaric levobupivacaine 20 ml epidurally with 0.5 ml distilled water.

Group B-30 patients were randomly assigned to receive 0.5% isobaric levobupivacaine 20 ml plus 0.5 ml dexmedetomidine containing 50 micrograms.

This study evaluated the following parameters like time of onset of sensory blockade at T10 level, maximum sensory blockade achieved and time taken to achieve the same, onset time of motor blockade, degree of motor blockade, time taken to achieve maximal motor blockade, hemodynamic changes in pulse rate, blood pressure and saturation, side effects and complications, intraoperative sedation scores, duration of analgesia, sensory & motor blockade, and any postoperative adverse reactions.

The data obtained from the above parameters in both the groups were statistically analysed using “statistical package for social sciences software” and p value of <0.05 was considered to be statistically significant. Demographic parameters

such as age, sex, weight and height have no statistically significant difference between the 2 groups.

There was statistically significant difference between the 2 groups in maximal sensory level achieved. In group A it ranged from T6 to T8 and in group B it ranged from T4 to T6.Time of onset of sensory level to T10 and the time taken to achieve the maximum level of sensory blockade was slightly lesser in group B compared to group A but showed no statistical significance.

Mean time of onset of motor blockade and maximum motor blockade achieved also showed statistically significant difference between the 2 groups with group A showing 19.33 minutes ranging from Bromage scale 1 to 2 as opposed to group B showing only 14.5 minutes ranging from Bromage scale 2 to 3.

Duration of analgesia, sensory and motor blockade were all found to be significantly higher in group B(477.3 minutes,568 minutes,265 minutes) as compared to group A(351.67 minutes,459.5 minutes,122.5 minutes)respectively.

Adverse effects experienced in general were statistically insignificant in both the groups. Mean sedation score in group B (Dexmedetomidine group) was predominantly found to be 2 as per Ramsay sedation score. None of the patients in group B had deep sedation or profound respiratory depression.

CONCLUSION

This study concludes that combining dexmedetomidine 50 micrograms with 0.5% isobaric levobupivacaine epidurally helps in achieving maximal sensory level of T4 to T6.Also the onset time of motor blockade was shortened with the addition of dexmedetomidine and the maximal motor blockade achieved was also intense with dexmedetomidine. Duration of analgesia, sensory and motor blockade were prolonged when levobupivacaine is combined with dexmedetomidine epidurally. Changes in hemodynamic parameters (blood pressure & heart rate) were very minimal in the dexmedetomidine group. Adverse effects encountered with dexmedetomidine were also acceptable with only mild discomfort to the patients. Absence of respiratory depression is a remarkable feature of dexmedetomidine.

Therefore combination of epidural dexmedetomidine with 0.5% isobaric levobupivacaine provides a better anaesthetic outcome in various aspects studied with minimal side effects than epidural 0.5% isobaric levobupivacaine alone.

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INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No. 19525/ME-1/Ethics/2013 Dt:03.01.2014
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Comparative Study of epidural 0.5% Levobupivacaine and epidural 0.5% Levobupivacaine with dexmedetomidine for patients undergoing elective infraumbilical and lower limb surgeries' – For Project Work Submitted by Dr.V.Mangal Swathi, MD (Anaesthesiology), PG Student, KMC, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

GOVT. KILPAUK MEDICAL COLLEGE * Kilpauk, Chennai-10. * 29 JAN 2014

CHAIRMAN, 29/1/14.
Ethical Committee
Govt.Kilpauk Medical College,
Chennai

PROFORMA

Name:

Age:

Sex:

IPno:

Group:

Date of admission: Date of surgery:

Preoperative examination:

Blood pressure:

Pulse rate :

Room air SpO2:

Cardiovascular system:

Respiratory system:

Central nervous system:

Diagnosis:

Surgery being performed: ASA:

Investigations:

Premedication:

Time of injection of study drug:

Time of onset of sensory block at T10 level:

Time of onset of motor blockade:

Maximum sensory blockade achieved & time taken:

Maximum motor blockade achieved (by modified Bromage scale):

Total duration of analgesia:

Total duration of sensory blockade:

Total duration of motor blockade:

Time of rescue analgesia & drug given:

Postoperative adverse effects if any:

INTRAOPERATIVE PARAMETERS:

Time Intervel in Minutes	HR	SpO2	SBP	DBP	MAP	Drug used
Basal						
1 min						
5						
10						
15						
20						
25						
30						
40						
50						
60						
70						
80						
90						
100						
110						
120						

	80 mins	90 mins	100 mins	110 mins	120 mins
HR					
SpO2					
SBP					
DBP					
MAP					
Drug used					

HR-Heart rate; SBP-Systolic blood pressure; DBP-Diastolic blood pressure;

MAP-Mean arterial pressure.

	Sedation score
Baseline	
30	
60	
90	
120	

PATIENT CONSENT FORM

Study detail : A Comparative study of epidural 0.5% isobaric Levobupivacaine and epidural 0.5% isobaric Levobupivacaine with Dexmedetomidine for patients undergoing elective infraumbilical and lower limb surgeries.

Study centre : GOVT. KILPAUK MEDICAL COLLEGE HOSPITAL & GOVT. ROYAPETTAH HOSPITAL , CHENNAI.

Patient's Name :

Patient's Age :

Identification Number :

Patient may check (☑) these boxes

- I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐ ☐
- I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.
- I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well-being or any unexpected or unusual symptoms. ☐ ☐
- I hereby consent to participate in this study.
- I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests. ☐

Signature/thumb impression:

Patients Name and Address:

Place: Date:

Signature of investigator :

Study investigator's Name :

சுய ஒப்புதல் படிவம்

A COMPARATIVE STUDY OF EPIDURAL 0.5% LEVOBUPIVACAINE AND EPIDURAL 0.5% LEVOBUPIVACAINE WITH DEXMEDETOMIDINE IN PATIENTS UNDERGOING ELECTIVE INFRAUMBILICAL AND LOWER LIMB SURGERIES.

ஆராய்சி நிலையம் : மயக்கவியல் துறை
கீழ்பாக்கம், மருத்துவக் கல்லூரி
சென்னை - 600 010

பங்கு பெறுபவரின் பெயர் : வயது :

பங்கு பெறுபவரின் எண். :

பங்கு பெறுபவரது இதனை (✓) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களைப் பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்தக் காரணத்தினாலலோ எந்தக் கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம்என்று அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்மந்தமாகவோ. இதைச் சார்ந்த மேலும் ஆய்வு மேற்கொள்ளம்போது இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளைப் பார்ப்பதற்கு என் அனுமதி தேவையில்லைஎன அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்துவிலகிக் கொண்டாலும் பொருந்தும் என் அகிறிறேன். ☐

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும். பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான முடிவுகளையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதைப் பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்குக் கூறப்பட்ட அறிவுரைக்ளின்படி நடந்து கொள்வதுடன், இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றம் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லது எதிர்பாராத நோய்க்குறி தென்ப்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன் ☐

பங்கேற்பவரின் கையொப்பம் ..... இடம் ..... தேதி

பங்கேற்பவரின் பெயர்மற்றும் விலாசம் .....

அய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி

ஆய்வாளிரின் பெயர் .....

MASTER CHART KEYWORDS

DUR	-	Duration
HR	-	Heart Rate
SBP	-	Systolic Blood Pressure
DBP	-	Diastolic Blood Pressure
MAP	-	Mean arterial Blood Pressure
SB	-	Sensory Blockade
MB	-	Motor Blockade
Analg	-	Analgesia
Sscore	-	Sedation Score
SpO_2	-	Oxygen Saturation

MASTER CHART

S.No	NAME	IP NO	AGE	SEX	GROUP	HEIGHT	WEIGHT	ASA	DIAGNOSIS	SURGERY DONE	Dur surgery	onset time to T10	Max sen Level	Time for max sens level	onset time MB	DEGREE MB	Max_time_MB	dur_analg	DUR_SB	dur_MB	Adverse effect
1	EGAMBARAM	23847	41	M	A	168	60	PS I	LT HYDROCELE WITH PHIMOSIS	LEFT SAC EVERSION WITH CIRCUMCISION	90	15	T8	20	20	BR 2	25	240	330	150	NIL
2	GOPALAKRISHNAN	23946	42	M	A	170	68	PS I	B/L INDIRECT INGUINAL HERNIA	B/L HERNIOPLASTY	100	12	T6	20	15	BR 3	25	390	480	120	NIL
3	DEVADARSAN	1409334	33	M	A	170	76	PS I	RT INGUINAL HERNIA	RIGHT HERNIOPLASTY	70	22	T6	30	30	BR 2	40	240	360	90	NIL
4	MUNNUSAMY	1409300	35	M	A	164	62	PS I	LT INGUINAL HERNIA WITH LTHYDROCELE	LEFT HERNIOPLASTY WITH LT SAC EVERSION	110	14	T6	22	25	BR 2	35	380	480	120	NIL
5	JAYAKODI	1410123	30	F	A	158	54	PS I	LEFT FEMORAL HERNIA	LEFT HERNIOPLASTY	120	6	T6	16	10	BR 3	20	370	450	150	NIL
6	DAMODARAN	1410784	40	M	A	164	62	PS I	B/L HYDROCELE	B/L SAC EVERSION	70	10	T6	14	15	BR 3	20	365	495	120	NIL
7	MEERA	1410892	43	F	A	155	54	PSII	INCISIONAL HERNIA	MESH REPAIR	70	8	T6	16	10	BR 3	20	240	375	120	NIL
8	LAKSHMI	1410906	35	F	A	154	52	PSI	UMBILICAL HERNIA	MESH REPAIR	100	14	T4	24	20	BR 2	35	420	540	90	URINARY RETENTION
9	BAKRUDDIN	1454725	38	M	A	166	60	PS I	LEFT HYDROCELE	LEFT SAC EVERSION	70	16	T6	22	20	BR 2	30	375	465	105	NIL
10	VADIVEL	1456452	40	M	A	160	58	PSII	RT INGUINAL HERNIA WITH RT HYDROCELE	RT HERNIOPLASTY WITH RT SACEVERSION	100	16	T6	26	25	BR 2	35	390	480	120	NIL
11	KAALI	1414564	28	M	A	162	64	PS I	SUBACUTE APPENDICITIS	OPEN APPENDICECTOMY	70	14	T4	22	20	BR 3	30	270	420	120	NIL
12	ARUL	1412462	36	M	A	166	62	PS I	INCISIONAL HERNIA	ANATOMICAL REPAIR	90	12	T6	20	15	BR 2	25	390	510	90	NIL
13	MOHAN	1414565	26	M	A	167	70	PS I	RAW AREA RT FOOT	SSG	100	10	T6	20	15	BR 2	20	330	420	150	NIL
14	JOTHI	1414567	32	F	A	165	60	PS I	INCISIONSL HERNIA	MESH REPAIR	90	16	T8	20	20	BR 3	25	300	450	120	NIL
15	SUNDARAM	1414602	32	M	A	169	67	PS I	B/L INDIRECT HERNIA	B/L HERNIOPLASTY	120	10	T8	16	15	BR 3	25	360	480	150	NIL
16	DINESH	1414539	29	M	A	169	66	PS I	AZOOSPERMIA	LT VARICOCELECTOMY	90	8	T6	16	20	BR 3	30	405	480	120	NIL
17	MAHESHWARI	1415474	32	F	A	160	62	PS I	RT FEMORAL HERNIA	RT HERNIORRHAPHY	70	12	T6	18	20	BR 3	20	375	510	150	NIL
18	ELUMALAI	1414567	40	M	A	166	58	PS II	LT HAEMATOCELE	LT SAC EVERSION WITH DRAINAGE	70	16	T6	26	25	BR 2	35	390	480	90	NIL

19	SAIRAM	1414752	39	M	A	165	63	PS I	LT INGUINAL HERNIA	LT HERNIO PLASTY	80	12	T6	16	20	BR 2	30	330	420	90	NIL
20	SEKAR	1450745	30	M	A	167	69	PS I	RT HYDROCELE	RT SAC EVERSION	70	14	T6	18	20	BR 2	25	270	420	90	NIL
21	SAMSON	1415247	32	M	A	166	64	PS I	RAW AREA LT LEG	SSG AND FLAP COVER	90	14	T6	20	20	BR 2	25	330	420	90	NAUSEA,VOMITING
22	MAHALAKSHMI	1414562	40	F	A	158	54	PS I	RT RECURRENT INGINAL HERNIA	RT HERNIOPLASTY	90	8	T8	16	15	BR 3	30	360	480	150	NIL
23	RAJESH	1415674	34	M	A	160	57	PS I	INCISIONAL HERNIA	MESH PLASTY	90	14	T8	20	20	BR 3	30	405	480	120	NIL
24	KASTHURI	1414567	38	F	A	164	62	PS I	SUBACUTE APPENDICITIS	OPEN APPENDICECTOMY	90	16	T6	20	20	BR 2	25	390	480	150	URINARY RETENTION
25	SAKTHIVEL	1414562	26	M	A	166	60	PS I	RT INGUINAL HEERNIA	RT HERNIOPLASTY	80	16	T8	22	20	BR 2	30	300	420	120	NIL
26	ANAND	1415672	34	M	A	163	64	PS I	LT HYDROCELE WITH PHIMOSS	LT SAC EVERSION WITH CIRCUMCISION	70	9	T6	20	20	BR 2	30	360	450	120	NIL
27	SUKUMAR	1413567	37	M	A	165	64	PS I	RT INGUINAL HERNIA WITH RT HYDROCELE	RT HERNIOPLASTY WITH RT SAC EVERSION	70	12	T6	16	25	BR 3	30	390	480	150	NIL
28	GANESH	1454765	31	M	A	167	64	PS I	B/L HYDROCELE	B/L SAC EVERSION	70	12	T6	20	20	BR 2	30	420	540	120	NIL
29	SAIRABANU	1414567	40	F	A	160	57	PSII	INCISIONAL HERNIA	ANATOMICAL REPAIR	90	6	T6	16	15	BR 2	20	405	510	180	NIL
30	GOPI	1414325	42	M	A	164	60	PS II	RECURRENT INCISIONAL HERINA	MESH REPAIR	80	10	T6	18	25	BR 2	35	360	480	120	URINARY RETENTION

Group B																									
S NO	NAME	IP NO	AGE	SEX	GROUP	HEIGHT	WEIGHT	ASA	DIAGNOSIS	SURGERY DONE	Dur surgery	onset time to T10	Max sen Level	Time for max sens level	onset time MB	DEGREE MB	Max_time_MB	dur_analg	DUR_SB	dur_MB	Adverse effect	SSCORE_B	SSCORE_30M	SSCORE_60M	SSCORE_90M	SSCORE_120M
1	BALAMURUGAN	5666	25	M	B	168	64	PS 1	LEFT VARICOSE VEINS	LEFT TRENDELENBERG PROCEDURE	80	12	T4	22	20	BR 2	30	405	540	240	DRY MOUTH	1	2	2	2	2
2	NIXON	1410154	32	M	B	166	59	PS 1	CHRONIC APPENDICITIS	OPEN APPENDICECTOMY	70	14	T4	26	15	BR 3	25	460	540	255	NIL	1	2	2	2	2
3	SURESH	1410168	28	M	B	165	66	PS 1	SUBACUTE APPENDICITIS	OPEN APPENDICECTOMY	70	12	T6	20	20	BR 3	30	480	555	280	NAUSEA ,VOMITING	1	2	2	2	2
4	MURUGAN	1411245	35	M	B	166	68	PS 1	RIGHT INGUINAL HERNIA	RIGHT HERNIOPLASTY	100	10	T6	22	20	BR 3	25	480	600	300	NIL	1	2	2	2	2
5	LAKSHMANAN	1411276	31	M	B	168	67	PS 1	RIGHT INGUINAL HERNIA WITH FISSURE IN ANO	RIGHT HERNIOPLASTY WITH LATERAL SPHINCTEROTOMY	110	12	T4	22	15	BR 3	25	420	510	315	NIL	1	2	2	2	2

6	RAVI	1412020	45	M	B	166	60	PS II	UMBILICAL HERNIA	ANATOMICAL REPAIR	70	8	T4	12	10	BR 3	15	525	600	240	NIL	1	1	2	2	2
7	THANUJA	14123452	36	F	B	156	52	PS I	INCISIONAL HERNIA	ANATOMICAL REPAIR	120	10	T4	20	15	BR 3	20	600	720	300	SHIVERING	1	1	1	2	2
8	GOWRI	1414068	40	F	B	162	59	PSI	INCISIONAL HERNIA	MESH REPAIR	60	8	T4	14	10	BR 3	15	480	570	270	NIL	1	2	2	2	2
9	SUKUMAR	1414542	26	M	B	167	54	PS I	RT VARICOSE VEINS	RT TRENDELENBERG OPERATION	120	10	T4	14	10	BR 2	15	420	495	300	NIL	1	2	2	2	2
10	SHANKAR	14`6478	32	M	B	166	64	PS I	POST TRAUMATIC RAW AREA RT LEG	FLAP COVER	80	12	T6	16	15	BR 2	15	450	540	270	NIL	1	2	2	2	1
11	SHANMUGAM	1401567	40	M	B	164	62	PS II	LT HYDROCELE WITH PHIMOSIS	LT SAC EVERSION WITH CIRCUMCISION	100	16	T6	22	15	BR 3	25	450	540	210	NIL	1	1	2	2	1
12	VENKATESAN	1416752	38	M	B	169	64	PS I	B/L INGUINAL HERNIA	B/L HERNIOPLASTY	100	22	T4	30	20	BR 2	30	420	570	270	SHIVERING	1	2	3	2	2
13	SHAKTHI	1412567	35	F	B	162	59	PS I	FEMORAL HERNIA	HERNIORRHAPHY	60	8	T4	16	15	BR 2	20	510	570	300	NIL	1	2	3	2	1
14	MALATHI	1410564	44	F	B	156	50	PS II	LIPOMA RT THIGH	EXCISION	80	10	T6	14	10	BR 3	25	480	540	270	NIL	1	1	2	2	1
15	VELMURUGAN	1424562	30	M	B	169	64	PS I	B/L HYDROCELE	B/L SAC EVERSION	60	12	T4	20	15	BR 3	20	510	600	300	NIL	1	2	3	2	1
16	SURENDRAN	1410462	34	M	B	169	62	PS I	B/L VARICOSE VEINS	B/L TRENDELENBERG PROCEDURE	135	14	T6	18	15	BR 3	25	465	540	330	NIL	1	2	3	2	2
17	SAKTHI	1415647	35	M	B	170	67	PS I	AZOOSPERMIA	RT VARICOCELECTOMY	80	10	T6	16	10	BR 3	20	540	600	300	NIL	1	2	2	2	1
18	KALPANA	1410562	27	F	B	162	54	PSI	LT RECURRENT INGUINAL HERNIA	LT MESH PLASTY	100	8	T6	12	10	BR 3	20	510	600	270	NIL	1	2	3	2	2
19	SEKAR	1414742	42	M	B	169	63	PS II	POST TRAUMATIC RAW AREA LT THIGH	SSG	90	8	T4	16	15	BR 3	20	540	630	270	NIL	1	2	2	2	2
20	PRABHU	1414562	40	M	B	166	60	PS II	LT INGUINAL HERNIA WITH B/L HYDROCELE	LT HERNIOPLASTY WITH B/L SAC EVERSION	90	12	T6	16	15	BR 3	20	480	540	300	NIL	1	2	2	2	2
21	NATHAN	1415672	39	M	B	167	62	PS I	LT TESTICILAR TUMOUR	LT ORCHIDECTOMY	100	6	T4	12	10	BR 3	15	525	600	210	NIL	1	2	2	2	2
22	SHAAHEERA	1415725	32	F	B	158	57	PS I	INCISIONAL HERNIA	ANATOMICAL REPAIR	110	10	T6	18	10	BR 3	20	465	510	180	NIL	1	2	2	3	2
23	RAJAN	1414562	28	M	B	168	64	PS I	SUBACUTE APPENDICITIS	OPEN APPENDICECTOMY	70	8	T4	18	15	BR 3	25	420	510	210	NIL	1	2	3	2	2
24	BABU	1414254	42	M	B	166	63	PS I	RT HYDROCELE WITH PHIMOSIS	RT SAC EVERSION WITH CIRCUMCISION	70	12	T6	10	15	BR 3	25	450	600	240	NIL	1	2	2	2	1
25	MARIMUTHU	1414567	32	M	B	166	59	PS I	RT INGUINAL HERNIA	RT HERNIOPLASTY	70	14	T4	20	20	BR 3	25	420	570	270	NIL	1	2	2	2	1
26	SANTHANAM	1414589	34	M	B	168	70	PS I	B/L HYDROCELE	B/L SAC EVERSION	70	14	T6	20	15	BR 3	25	495	600	240	NIL	1	2	3	2	1
27	SHANTHI	1414752	35	F	B	164	59	PSI	CHRONIC APPENDICITIS	OPEN APPENDICECTOMY	70	12	T6	16	15	BR 3	20	510	570	270	NIL	1	2	2	2	2
28	SRINIVASAN	1414754	34	M	B	170	68	PSI	LTVARICOSE VEINS	LT TRENDELENBERG PROCEDURE	100	14	T4	20	15	BR 3	25	480	570	270	NIL	1	2	3	2	1
29	SHANKARI	1414562	39	F	B	160	56	PSI	INCISIONAL HERNIA	MESH PLASTY	90	14	T4	20	10	BR 3	20	450	540	240	NIL	1	2	3	2	1
30	MUTHUKARUPPAN	1415647	29	M	B	169	60	PSI	UMBILICAL HERNIA	ANATOMICAL REPAIR WITH UMBILICECTOMY	70	10	T4	20	20	BR 2	25	495	570	240	NIL	1	2	2	2	2

INTRAOPERATIVE PARAMETERS

Patient	GROUP A	HR_base	HR_1m	HR_5m	HR_10m	HR_15m	HR_20 m	HR_25 m	HR_30 m	HR_40 m	HR_50m	HR_60m	HR_70m	HR_80 m	HR_90m	HR_100m	HR_110m	HR_120m	SB_base	SB_1m	SB_5m	SB_10m	SB_15m	SB_20 m	SB_25 m	SB_30 m	SB_40 m	SB_50m	SB_60m	SB_70m	SB_80 m	SB_90m	SB_100m	SB_110m	SB_120m
1	EGAMBARAM	90	86	82	78	76	78	74	72	76	76	74	70	72	74	76	74	72	140	142	138	122	124	122	122	134	140	142	132	136	132	140	130	132	134
2	GOPALAKRISHNAN	72	74	68	66	64	60	64	68	64	66	60	62	62	64	66	68	64	130	128	120	110	108	108	118	118	118	120	122	118	120	118	122	124	126
3	DEVADARSAN	78	80	74	72	72	70	72	70	66	66	68	64	64	64	66	66	64	130	132	128	124	120	118	122	118	120	114	112	114	124	120	118	116	122
4	MUNNUSAMY	78	82	74	74	72	68	64	62	60	62	64	68	60	64	62	62	64	120	122	130	110	108	114	92	88/E6	110	106	110	112	116	114	112	118	120
5	JAYAKODI	64	62	64	68	62	66	66	68	66	58	56	62	68	70	70	72	68	102	102	100	102	100	100	102	106	104	104	80/E6	102	94	112	110	114	106
6	DAMODARAN	78	80	74	72	74	70	68	66	62	64	64	68	66	64	68	66	72	140	136	142	130	132	140	142	138	132	134	136	134	130	134	132	134	134
7	MEERA	80	86	84	78	76	76	74	72	72	74	74	70	76	74	76	72	70	120	124	118	114	110	104	106	108	100	98	96	104	100	102	104	104	102
8	LAKSHMI	80	84	78	82	76	72	68	64	62	68	72	74	76	76	78	78	76	130	128	114	110	110	104	102	100	100	100	100	104	102	104	104	106	104
9	BAKRUDDIN	84	88	84	86	80	78	78	76	74	76	74	78	80	82	84	82	80	120	120	120	118	113	112	112	110	112	110	108	110	108	108	110	112	110
10	VADIVEL	84	88	86	84	80	78	76	80	78	78	74	72	68	70	68	68	70	132	130	128	124	122	118	116	114	110	112	110	112	110	112	110	1118	116
11	KAALI	90	94	92	88	86	84	80	82	80	82	78	78	76	76	78	78	76	118	120	122	120	122	118	112	110	108	108	106	108	112	110	114	112	114
12	ARUL	84	88	82	80	78	80	76	78	80	76	74	74	76	74	72	74	75	112	110	110	112	110	108	106	108	110	108	106	104	104	102	106	104	106
13	MOHAN	82	86	82	80	82	76	76	78	74	72	70	74	72	70	70	70	74	114	110	108	106	104	102	100	102	102	104	108	108	106	108	104	102	108
14	JOTHI	86	84	80	82	80	80	78	74	70	76	74	70	70	72	76	78	76	124	122	120	118	116	112	110	112	110	108	110	110	108	110	112	110	106
15	SUNDARAM	80	86	82	78	78	80	76	74	74	76	72	74	70	70	72	68	72	110	110	108	106	106	104	104	106	106	104	102	102	106	102	102	102	104
16	DINESH	86	88	84	82	80	78	78	76	80	76	82	78	72	70	72	74	76	130	130	128	124	122	122	120	118	116	114	112	110	108	106	108	110	112
17	MAHESHWARI	88	90	86	88	82	80	82	80	84	78	76	78	78	76	76	76	78	124	118	116	114	112	110	110	108	108	110	110	110	114	112	116	118	110
18	ELUMALAI	86	90	85	84	80	82	78	80	76	74	70	72	72	76	76	80	82	124	122	120	120	112	110	108	110	108	106	108	106	104	106	104	108	106
19	SAIRAM	82	88	84	82	86	78	82	76	74	72	70	72	74	76	78	76	78	110	112	110	108	108	106	108	110	106	104	100	100	102	100	104	106	106
20	SEKAR	94	96	90	88	92	86	84	80	82	80	82	80	84	86	88	86	84	120	118	116	114	110	112	108	106	108	106	104	102	108	112	106	110	112
21	SAMSON	92	96	98	90	86	88	84	80	82	80	76	78	76	74	76	76	78	114	116	114	112	110	108	106	104	104	102	104	102	102	104	110	112	110
22	MAHALAKSHMI	90	88	86	88	84	80	74	70	72	70	68	70	70	72	74	76	78	118	120	122	120	116	116	112	110	106	104	106	102	100	102	108	106	108
23	RAJESH	80	82	76	78	70	74	76	70	68	64	66	70	72	70	78	76	78	110	114	112	108	106	104	102	102	98	96	100	100	102	104	106	108	106
24	KASTHURI	90	94	92	88	86	80	82	82	84	80	78	76	78	76	78	76	76	122	120	118	116	110	112	108	106	110	110	108	106	108	106	114	120	118
25	SAKTHIVEL	86	90	88	84	82	80	82	78	76	74	70	74	72	80	78	78	76	130	128	124	122	120	118	112	110	110	112	108	110	110	112	114	112	110
26	ANAND	84	88	86	80	82	78	74	76	72	70	72	70	72	73	76	76	80	130	128	124	118	116	114	112	110	110	108	108	106	108	106	104	104	112
27	SUKUMAR	86	88	90	84	80	82	80	82	76	78	74	70	74	76	78	78	76	120	122	118	114	112	110	108	108	106	108	108	106	110	112	114	120	122
28	GANESH	88	92	90	86	84	82	80	76	74	70	68	70	72	76	78	76	82	124	122	120	118	116	114	112	110	108	106	108	106	110	112	110	120	122
29	SAIRABANU	92	96	90	92	88	86	80	82	78	80	76	74	76	74	78	84	82	140	138	134	130	128	128	124	130	128	124	122	120	122	120	124	126	126
30	GOPI	78	84	82	80	76	76	80	74	70	72	68	66	70	72	74	76	78	120	118	112	110	112	108	110	108	110	106	102	100	102	104	106	104	108
	GROUP B																																		
1	BALAMURUGAN	92	88	84	78	72	66	62	68	70	76	72	70	78	78	76	78	76	132	138	136	132	136	138	134	142	134	132	124	138	142	132	130	128	126
2	NIXON	88	82	76	70	60	56	54	56	54	58	60	60	64	62	64	68	72	132	140	136	140	130	128	126	124	122	120	122	120	122	124	126	122	130
3	SURESH	92	94	86	78	64	62	66	64	66	64	62	60	62	66	64	64	66	132	130	132	128	124	122	122	118	120	124	124	122	126	126	124	122	122
4	MURUGAN	82	86	80	78	72	66	62	56	52	48/A0.6	52	56	60	62	64	64	64	124	126	118	116	118	110	108	110	102	100	104	108	118	120	120	120	118
5	LAKSHMANAN	86	90	92	84	78	72	68	66	64	62	64	66	68	70	68	72	74	122	120	124	120	122	114	116	120	118	120	114	118	120	122	126	124	126
6	RAVI	98	102	92	88	82	82	80	78	74	70	72	70	72	74	76	74	74	130	120	110	114	110	100	90	110	114	108	104	106	110	112	114	110	112
7	THANUJA	98	96	92	88	84	78	74	72	66	64	62	66	60	62	64	62	66	120	124	114	110	104	100	94	92	92	96	104	102	100	100	102	104	102
8	GOWRI	112	104	100	96	94	90	88	84	84	86	84	84	84	86	84	80	82	130	120	96	80/E6	90	92	94	100	102	110	102	102	98	98	100	102	100
9	SUKUMAR	86	90	84	82	78	76	72	70	68	64	64	62	62	58	60	62	60	124	120	118	112	110	106	108	100	92	88	96	100	102	104	108	104	104
10	SHANKAR	80	84	62	80	74	74	70	70	64	56	56	58	74	76	72	74	74	122	126	120	114	112	110	100	102	92	96	96	100	102	108	106	98	104
11	SHANMUGAM	80	82	78	72	68	64	62	56	52	48	60	62	62	64	60	62	64	112	114	110	98	94	92	86/E6	98	100	90	96	102	104	102	102	106	108
12	VENKATESAN	90	92	94	88	82	76	72	68	70	66	68	64	66	62	64	66	64	120	124	118	108	104	102	98	92	90	100	102	104	102	108	110	112	108
13	SHAKTHI	78	84	80	74	72	68	84	58	50	56	58	60	60	64	62	64	64	130	122	110	108	110	102	100	100	102	96	96	98	100	104	106	102	112
14	MALATHI	94	98	90	86	80	80	62	76	74	72	70	68	64	66	66	68	72	110	112	108	110	102	104	98	96	92	90	100	102	100	104	106	108	110
15	VELMURUGAN	86	90	88	86	80	82	76	74	68	62	60	62	64	66	64	65	66	124	118	110	112	100	104	102	106	98	92	104	106	108	104	104	102	104
16	SURENDRAN	90	94	98	90	86	82	78	74	70	62	64	60	62	60	64	58	62	120	4	112	106	102	96	90	92	94	102	104	106	104	102	104	100	100
17	SAKTHI	78	82	80	74	72	72	70	64	62	56	56	58	56	60	62	64	68	120	118	112	108	106	98	92	92	90	94	98	100	102	100	102	98	104
18	KALPANA	86	90	88	84	80	82	76	70	64	62	60	60	58	60	62	66	64	110	116	108	106	98	96	90	92	84	90	108	104	102	100	104	106	108
19	SEKAR	92	96	90	88	86	84	78	70	64	60	58	56	60	58	62	65	64	130	128	118	110	100	98	92	94	92	90	84/E6	96	102	100	104	106	108
20	PRABHU	80	86	90	82	76	74	70	62	54	54	48/A.06	58	60	62	60	62	64	124	118	110	102	98	90	96	94	98	96	94	100	102	102	104	106	106
21	NATHAN	78	84	80	72	64	62	60	58	56	54	54	56	54	54	56	58	62	118	120	112	108	102	98	100	102	98	100	100	102	102	102	104	106	106
22	SHAAHEERA	88	94	86	84	84	86	80	76	70	64	66	68	64	62	60	62	64	118	120	110	108	106	104	102	98	96	94	96	92	90	94	98	98	102
23	RAJAN	96	98	92	88	86	82	80	78	70	68	64	62	64	64	66	66	68	120	120	118	116	114	108	106	102	104	102	100	102	104	106	108	110	112
24	BABU	86	84	88	80	76	72	72	68	62	60	58	58	60	62	64	64	66	110	112	108	104	108	106	108	102	98	96	94	96	98	98	108	110	108
25	MARIMUTHU	90	98	102	94	92	86	82	76	70	64	68	62	70	70	72	74	75	122	118	110	108	108	106	104	102	108	110	110	106	108	108	110	104	112
26	SANTHANAM	80	84	88	80	74	72	70	66	64	58	58	60	64	66	72	74	74	124	122	116	110	110	108	106	104	106	102	100	98	100	100	110	112	110
27	SHANTHI	86	92	86	84	80	78	82	74	68	62	60	64	66	68	66	66	68	112	114	110	108	108	104	104	102	102	100	102	102	106	108	110	114	112
28	SRINIVASAN	84	88	86	80	76	74	70	66	68	62	58	60	60	62	60	62	66	112	114	110	108	104	102	98	92	90	92	90	86	98	102	102	106	108
29	SHANKARI	82	84	86	80	82	80	76	70	68	68	64	62	60	58	68	68	70	120	120	120	118	112	110	108	106	106	102	102	100	98	102	108	110	118
30	MUTHUKARIPPAN	78	82	84	80	76	74	70	70	68	64	62	64	68	74	78	80	82	130	128	124	122	110	110	108	106	102	100	102	98	104	104	108	110	112

Patient	GROUP A	DB_base	DB_1m	DB_5m	DB_10m	DB_15m	DB_20 m	DB_25 m	DB_30 m	DB_40 m	DB_50m	DB_60m	DB_70m	DB_80 m	DB_90m	DB_100m	DB_110m	DB_120m	MAP_base	MAP_1m	MAP_5m	MAP_10m	MAP_15m	MAP_20 m	MAP_25 m	MAP_30 m	MAP_40 m	MAP_50m	MAP_60m	MAP_70m	MAP_80 m	MAP_90m	MAP_100m	MAP_110m
1	EGAMBARAM	80	72	76	86	78	86	82	86	82	84	84	88	84	86	84	84	86	100	97	97	98	93	98	95	102	101	103	100	98	100	104	98	96
2	GOPALAKRISHNAN	80	78	70	70	68	62	76	64	76	80	80	78	80	68	74	78	78	97	95	87	83	81	77	90	82	90	93	94	91	93	85	90	92
3	DEVADARSAN	76	80	74	70	82	76	78	82	68	70	80	72	74	74	76	78	78	94	97	92	88	95	90	93	94	85	85	91	86	88	84	82	84
4	MUNNUSAMY	70	72	74	68	64	60	56	52	70	70	68	62	70	72	76	70	72	87	89	93	82	79	78	68	64	83	82	82	79	85	86	88	86
5	JAYAKODI	72	70	62	74	68	62	64	76	68	68	54	64	68	60	60	70	68	82	81	75	83	79	75	77	86	80	80	63	77	77	77	80	85
6	DAMODARAN	82	86	80	70	68	70	76	62	70	68	62	64	66	68	68	66	68	101	103	101	90	89	93	98	87	91	90	87	88	86	84	86	88
7	MEERA	70	72	68	68	62	60	62	64	56	54	52	62	62	62	62	68	64	87	89	85	83	78	75	77	79	71	69	67	76	75	75	76	80
8	LAKSHMI	70	72	68	70	68	62	64	62	68	70	72	72	72	76	72	74	76	90	91	83	83	82	76	77	75	79	80	85	83	82	85	83	84
9	BAKRUDDIN	72	74	78	74	72	68	68	70	72	74	72	70	72	72	74	76	78	88	90	92	89	87	83	83	83	85	86	84	83	82	80	78	84
10	VADIVEL	70	68	70	72	68	70	72	74	64	68	70	70	68	64	72	74	76	91	89	89	89	86	86	87	87	79	83	83	84	82	80	85	84
11	KAALI	74	74	70	68	64	66	62	60	64	62	60	64	66	64	72	70	72	89	89	87	85	83	83	79	77	79	77	75	79	80	82	83	82
12	ARUL	80	78	82	80	70	74	76	70	70	68	62	66	66	68	70	72	74	91	89	91	91	83	85	86	83	83	81	77	79	79	79	80	82
13	MOHAN	82	84	78	74	72	74	68	64	66	64	66	64	66	68	64	68	76	93	93	88	85	83	83	79	77	78	77	80	79	79	81	77	80
14	JOTHI	80	82	76	72	70	72	68	64	62	62	60	62	58	60	64	66	68	95	95	91	87	85	85	82	80	78	77	77	78	75	77	76	78
15	SUNDARAM	62	64	62	64	68	62	68	62	64	60	60	62	64	60	60	56	60	78	79	77	78	81	76	80	77	78	75	74	75	78	74	74	71
16	DINESH	80	78	74	72	68	64	68	70	72	68	62	64	70	62	64	66	68	97	95	92	89	86	83	85	86	87	83	79	79	83	77	78	80
17	MAHESHWARI	72	70	68	70	68	62	60	60	56	60	62	60	64	68	70	72	76	89	86	84	85	83	78	77	76	73	77	78	77	78	80	82	84
18	ELUMALAI	78	74	76	78	70	68	62	64	62	68	70	72	76	72	70	72	74	93	90	91	92	84	82	77	79	77	81	83	83	78	80	82	78
19	SAIRAM	68	70	72	64	62	64	68	62	56	52	50	54	58	62	64	66	70	82	84	85	79	77	78	81	78	73	69	67	69	73	74	76	78
20	SEKAR	82	80	78	72	78	80	76	72	70	68	64	66	72	74	78	76	74	95	93	91	86	89	91	87	83	83	81	77	78	77	78	80	82
21	SAMSON	82	84	80	78	74	70	68	68	66	64	62	60	64	66	68	66	68	93	95	91	89	86	83	81	80	79	77	76	74	77	79	80	82
22	MAHALAKSHMI	76	74	72	68	64	66	62	60	56	54	56	52	60	62	64	66	68	90	89	89	85	81	83	79	77	73	71	73	69	73	75	78	78
23	RAJESH	84	86	70	64	62	60	62	60	56	54	56	58	54	56	62	64	66	93	95	84	79	77	75	75	74	70	68	71	72	70	72	72	76
24	KASTHURI	70	60	62	60	60	58	56	52	54	56	60	62	66	62	64	68	66	87	85	81	79	77	76	73	70	73	74	76	77	80	77	78	78
25	SAKTHIVEL	76	76	70	68	64	62	64	66	64	60	60	62	60	62	64	68	70	94	93	88	86	83	81	80	81	79	77	76	78	77	78	76	74
26	ANAND	62	64	60	58	56	56	58	52	52	50	54	54	58	56	58	60	62	85	85	81	78	76	75	76	71	71	69	72	71	76	78	74	76
27	SUKUMAR	74	76	70	68	62	60	60	62	64	62	60	62	64	66	68	64	66	89	91	86	83	79	77	76	77	78	77	76	77	76	78	79	79
28	GANESH	82	80	76	72	70	68	62	60	56	62	64	52	56	58	60	62	64	96	94	91	87	85	83	79	77	73	77	79	77	78	82	83	84
29	SAIRABANU	70	72	68	70	64	68	66	62	60	62	64	64	66	68	72	70	74	96	94	90	90	85	88	85	65	83	83	83	83	85	85	84	80
30	GOPI	70	72	68	64	62	60	56	54	58	56	60	60	54	58	62	66	68	87	87	83	79	79	76	74	72	75	73	74	73	70	72	74	76
	GROUP B																																	
1	BALAMURUGAN	84	82	78	72	76	84	74	84	86	84	88	84	74	78	76	80	76	103	101	97	92	96	102	94	100	102	100	100	102	97	96	94	92
2	NIXON	78	82	78	72	62	68	64	62	68	66	68	64	66	64	62	70	74	96	101	97	95	85	88	85	83	86	84	86	83	86	86	88	84
3	SURESH	78	84	76	80	72	70	80	82	86	78	78	68	70	72	76	74	74	96	99	95	96	89	87	94	94	97	93	93	86	88	90	86	90
4	MURUGAN	72	82	76	64	62	60	60	56	58	60	60	62	66	68	70	72	74	89	97	90	81	81	77	76	74	73	73	75	77	83	85	89	78
5	LAKSHMANAN	84	86	78	70	68	76	60	62	76	64	68	62	72	78	82	84	80	97	97	93	87	86	89	79	81	90	83	63	81	88	96	97	97
6	RAVI	70	84	76	72	68	64	60	70	62	72	70	72	74	76	74	72	70	90	96	87	86	82	76	70	83	79	84	81	83	84	85	86	86
7	THANUJA	72	70	68	60	56	52	50	52	54	56	60	64	68	70	64	64	64	88	88	83	77	72	68	65	64	67	69	75	77	79	80	77	77
8	GOWRI	72	62	60	50	54	56	60	60	60	64	60	56	58	60	64	66	62	91	81	72	60	66	68	71	73	74	79	74	71	74	76	78	76
9	SUKUMAR	76	68	64	62	56	56	52	56	52	50	56	60	62	64	64	66	68	92	85	82	79	74	73	71	71	65	63	69	73	75	77	79	79
10	SHANKAR	68	70	68	62	60	56	62	64	48	60	62	60	56	58	62	66	64	89	89	85	79	77	74	75	77	63	72	73	73	74	76	78	77
11	SHANMUGAM	84	76	68	62	56	60	52	60	60	58	60	62	68	70	68	72	68	93	89	82	74	69	71	63	73	73	69	72	75	80	81	79	78
12	VENKATESAN	68	70	58	56	52	50	52	52	50	56	60	60	64	66	62	64	64	89	88	78	73	69	67	67	65	63	71	74	75	77	80	78	79
13	SHAKTHI	70	60	56	58	60	56	54	54	58	50	52	54	56	62	64	66	68	90	81	74	75	77	71	69	69	73	65	65	68	72	74	76	75
14	MALATHI	60	68	60	54	56	56	58	52	54	56	62	58	62	64	66	64	66	77	83	76	69	71	72	71	67	67	67	75	73	75	70	72	71
15	VELMURUGAN	82	76	62	56	58	60	64	68	70	50	58	64	66	68	70	72	74	96	90	78	71	72	75	77	81	79	64	73	74	76	78	77	70
16	SURENDRAN	76	68	60	56	52	50	52	54	52	58	62	60	68	62	64	60	58	91	84	77	73	69	65	65	67	63	73	76	75	80	75	77	73
17	SAKTHI	78	74	68	64	66	62	64	60	54	58	64	62	60	64	64	66	68	92	89	83	79	79	74	73	71	65	70	75	75	74	72	70	68
18	KALPANA	84	78	72	70	64	68	62	66	58	52	60	58	60	58	64	66	68	93	91	84	82	75	77	71	75	67	65	76	73	74	72	77	78
19	SEKAR	80	82	80	76	64	62	58	62	64	66	60	70	70	68	70	74	70	97	97	93	87	76	74	69	73	73	74	68	79	81	79	74	77
20	PRABHU	68	62	56	52	50	48	60	58	54	60	64	62	66	64	66	68	64	87	81	74	69	66	62	72	70	65	71	74	75	78	77	72	74
21	NATHAN	72	70	62	60	56	58	60	64	62	56	54	58	60	60	62	64	66	87	87	79	76	71	71	73	77	74	71	69	73	60	60	62	66
22	SHAAHEERA	72	72	68	68	70	62	58	52	50	52	56	54	56	60	62	62	64	87	88	82	81	82	76	73	67	65	66	69	67	67	71	74	74
23	RAJAN	68	64	62	60	56	56	60	58	52	54	56	60	62	62	64	66	68	85	83	81	79	75	73	75	73	69	70	71	74	76	77	76	76
24	BABU	74	70	68	62	64	62	64	60	54	52	52	56	60	62	64	66	64	86	84	81	76	79	77	79	74	69	67	66	69	72	75	76	78
25	MARIMUTHU	74	72	70	70	68	62	60	58	54	56	52	54	56	58	60	62	64	90	87	83	83	81	77	75	73	72	74	71	71	72	74	74	74
26	SANTHANAM	78	72	68	62	64	68	70	62	68	62	60	64	66	68	68	70	68	93	89	84	78	79	81	82	76	81	75	73	75	75	74	73	70
27	SHANTHI	70	68	60	60	62	60	62	60	56	58	64	66	62	64	66	66	68	84	83	77	76	77	75	76	74	71	72	77	78	74	75	76	75
28	SRINIVASAN	70	70	68	62	56	52	50	50	52	56	58	60	64	60	62	58	62	84	85	82	77	72	69	66	64	65	68	69	72	75	74	75	70
29	SHANKARI	86	84	76	74	70	68	62	56	56	52	58	60	62	54	56	58	62	97	96	91	89	84	82	77	73	73	69	73	73	74	70	74	73
30	MUTHUKARIPPAN	68	70	64	72	68	64	64	66	62	60	60	62	64	64	66	64	62	89	89	84	89	82	79	79	79	75	73	74	74	75	76	81	80

Patient	GROUP A	MAP_120m	SpO2 base	SpO2 1m	SpO2 5m	SpO2 10m	SpO2 15M	SpO2 20m	SpO2 25m	SpO2 30m	SpO2 40m	SpO2 50m	SpO2 60m	SpO2 70m	SpO2 80m	SpO2 90m	SpO2 100	SpO2 110	SpO2 120
1	EGAMBARAM	94	98%	98%	98	99	98	98	99	100	99	99	99	98	99	98	99	99	98
2	GOPALAKRISHNAN	90	99	100	99	99	98	99	99	99	99	99	99	98	99	98	99	98	99
3	DEVADARSAN	88	100	99	99	100	99	99	99	99	98	99	100	99	100	99	98	99	100
4	MUNNUSAMY	84	99	98	99	98	99	99	99	98	98	98	98	99	99	99	99	99	99
5	JAYAKODI	81	98	98	99	99	98	99	98	99	98	98	98	99	98	99	98	98	98
6	DAMODARAN	86	99	99	99	98	99	98	98	98	98	98	98	99	99	98	98	98	98
7	MEERA	77	100	99	100	99	100	100	99	99	99	98	99	100	99	99	99	99	99
8	LAKSHMI	84	98	98	98	98	98	98	98	98	98	98	99	99	98	98	98	98	99
9	BAKRUDDIN	85	99	98	99	98	99	98	99	98	99	98	99	98	99	99	99	98	99
10	VADIVEL	86	100	100	99	100	100	99	100	100	99	99	98	99	100	99	100	99	100
11	KAALI	78	99	98	99	98	99	98	99	99	99	99	100	99	100	99	100	99	99
12	ARUL	86	99	98	99	99	99	99	99	99	99	99	99	99	99	98	99	98	99
13	MOHAN	82	98	99	99	98	99	98	98	99	98	99	98	99	99	98	98	98	99
14	JOTHI	80	99	98	99	99	99	99	99	99	99	98	99	99	99	98	99	99	98
15	SUNDARAM	75	99	99	98	98	98	98	98	99	99	99	98	98	99	99	98	99	99
16	DINESH	82	98	98	98	98	99	98	99	98	99	98	99	98	99	98	98	98	98
17	MAHESHWARI	80	98	99	98	99	98	98	99	98	98	99	98	99	98	99	98	99	98
18	ELUMALAI	80	99	98	98	98	98	99	98	98	98	98	98	98	98	98	98	99	99
19	SAIRAM	77	98	99	98	99	98	98	99	98	98	98	99	98	98	98	98	98	99
20	SEKAR	84	99	99	99	98	98	99	98	99	98	99	98	99	99	99	99	98	99
21	SAMSON	84	98	98	99	98	99	98	99	98	98	98	98	99	98	99	98	99	98
22	MAHALAKSHMI	80	99	100	99	100	100	99	100	99	100	99	99	99	100	100	99	100	99
23	RAJESH	74	98	99	99	99	98	99	99	100	99	98	98	99	98	99	98	99	99
24	KASTHURI	80	98	99	99	99	99	98	99	99	99	98	99	98	99	98	99	99	98
25	SAKTHIVEL	72	98	99	98	99	98	99	99	98	98	99	98	98	99	99	99	98	98
26	ANAND	78	99	99	99	98	98	99	98	99	99	98	98	99	99	98	99	98	99
27	SUKUMAR	82	98	99	99	99	98	98	99	98	99	98	99	99	99	98	99	98	98
28	GANESH	80	99	99	99	99	99	99	98	99	98	98	99	98	99	98	99	98	99
29	SAIRABANU	80	98	99	98	99	98	99	98	99	98	99	98	99	98	98	98	98	98
30	GOPI	76	99	98	98	99	98	99	98	98	98	98	98	98	98	98	98	98	98
	GROUP B																		
1	BALAMURUGAN	90	99	99	99	98	98	98	99	98	99	98	98	98	98	98	98	98	99
2	NIXON	84	98	99	98	98	99	99	99	98	99	99	98	99	98	99	98	99	98
3	SURESH	88	99	98	99	98	99	98	98	99	99	99	98	98	99	98	99	98	99
4	MURUGAN	80	98	99	99	98	98	98	99	99	98	99	98	99	98	98	99	98	99
5	LAKSHMANAN	88	98	99	98	99	98	99	98	99	98	98	98	99	98	98	98	98	99
6	RAVI	86	99	99	99	98	99	99	98	99	98	98	99	99	99	99	99	99	98
7	THANUJA	77	99	98	98	98	99	98	99	98	99	98	98	98	99	98	98	98	98
8	GOWRI	74	98	99	98	99	98	98	98	98	99	98	99	98	98	98	98	98	99
9	SUKUMAR	80	99	98	98	99	99	98	99	98	99	98	99	98	98	99	98	99	99
10	SHANKAR	74	98	98	98	99	98	99	98	99	98	99	98	98	98	98	98	99	98
11	SHANMUGAM	76	99	99	98	99	98	99	98	99	98	98	99	98	98	99	98	99	98
12	VENKATESAN	80	98	98	99	98	99	98	99	98	99	99	99	98	99	98	98	99	99
13	SHAKTHI	74	99	99	98	98	99	99	99	98	99	98	99	98	99	99	98	98	98
14	MALATHI	74	100	100	99	99	99	98	99	98	99	100	99	99	100	99	99	98	99
15	VELMURUGAN	68	99	99	99	98	99	98	99	98	99	98	99	98	99	98	99	99	98
16	SURENDRAN	72	99	98	99	98	99	98	99	99	99	98	99	98	99	99	98	98	98
17	SAKTHI	70	99	99	99	98	99	99	98	99	98	99	98	99	99	98	99	98	99
18	KALPANA	76	99	99	98	98	98	99	98	98	98	98	98	98	98	98	98	98	99
19	SEKAR	76	98	98	98	98	99	98	98	98	99	99	99	98	98	99	98	99	98
20	PRABHU	72	99	99	99	99	99	99	99	99	99	99	99	99	99	99	99	99	99
21	NATHAN	64	99	98	99	98	99	99	98	99	98	99	98	99	99	99	99	99	98
22	SHAAHEERA	75	98	98	98	98	99	98	99	98	99	98	99	98	99	98	99	98	98
23	RAJAN	78	98	98	98	98	98	98	98	98	98	98	98	98	98	98	98	98	98
24	BABU	74	98	99	99	99	98	98	98	99	98	99	98	99	98	99	98	99	98
25	MARIMUTHU	70	98	99	99	99	98	99	98	99	98	99	98	98	98	99	99	98	99
26	SANTHANAM	78	99	98	99	98	99	98	98	98	99	99	98	99	98	99	98	98	98
27	SHANTHI	72	98	99	99	98	99	98	99	98	99	98	99	98	99	98	99	98	99
28	SRINIVASAN	68	99	99	99	99	98	99	98	99	98	99	99	98	98	98	98	99	98
29	SHANKARI	74	98	99	98	99	98	99	99	99	98	98	99	98	99	98	99	98	98
30	MUTHUKARIPPAN	78	99	99	98	98	98	99	98	99	98	98	98	98	99	99	98	98	98

POST OPERATIVE PARAMETERS

Patient	GROUP A	HR 3H	HR 4H	HR 5H	HR 6H	HR 8H	HR10 H	HR 12H	SBP 3H	SBP 4H	SBP 5H	SBP 6H	SBP 8H	SBP 10 H	SBP 12H	DBP 3H	DBP 4H	DBP 5H	DBP 6H	DBP 8H	DBP 10	DBP 12	MAP 3H	MAP 4H	MAP 5H	MAP 6H	MAP 8H	MAP 10H	MAP 12H	SPO2 3H	SPO2 4H	SPO2 5H	SPO2 6H	SPO2 8H	SPO2 10 H	SPO2 12H
1	EGAMBARAM	78	78	76	80	82	84	88	110	114	112	118	116	110	112	64	62	64	70	72	74	76	79	79	80	86	86	86	88	99	98	99	98	99	99	98
2	GOPALAKRISHNAN	70	74	76	78	78	80	84	120	116	122	114	120	122	124	70	72	74	76	78	76	78	87	87	90	89	92	91	93	99	98	99	99	98	99	99
3	DEVADARSAN	80	82	84	84	86	82	84	130	132	124	128	126	120	122	68	66	64	70	72	74	76	89	88	84	89	90	89	91	98	98	99	98	99	98	98
4	MUNNUSAMY	78	78	84	86	80	84	88	110	110	108	106	108	108	112	70	72	74	76	78	78	76	83	85	85	86	88	88	88	98	98	98	98	99	98	99
5	JAYAKODI	80	80	82	84	86	86	84	120	122	124	122	120	110	118	66	68	66	70	74	76	74	84	86	85	87	89	87	89	99	99	98	99	98	99	98
6	DAMODARAN	78	76	76	78	78	84	86	116	120	122	124	126	126	120	78	82	84	82	82	80	82	91	95	96	96	97	97	95	98	98	99	99	98	99	98
7	MEERA	76	78	80	78	82	82	80	126	128	124	122	120	124	126	76	76	78	80	84	82	80	93	93	93	94	96	96	95	99	98	98	98	99	99	98
8	LAKSHMI	78	78	78	82	84	84	86	120	116	114	110	110	116	124	82	84	86	86	84	86	84	95	95	95	94	93	96	97	99	98	98	98	99	98	99
9	BAKRUDDIN	80	80	82	84	86	88	86	100	108	110	106	106	108	108	68	64	70	72	68	66	66	79	79	83	83	81	80	80	97	98	98	99	98	99	99
10	VADIVEL	68	70	76	76	78	80	82	102	110	112	110	120	122	124	76	78	82	84	84	84	86	85	89	92	93	96	97	99	100	100	99	100	99	98	99
11	KAALI	70	70	72	74	76	78	80	106	106	108	104	104	102	112	64	62	62	66	68	68	70	78	77	77	79	80	79	84	100	100	99	98	99	99	99
12	ARUL	72	70	72	74	76	78	78	120	122	124	126	128	124	126	80	78	76	76	80	74	74	93	93	92	93	96	91	91	99	99	100	100	99	99	100
13	MOHAN	80	82	84	86	84	84	86	114	116	110	112	110	112	116	70	70	68	72	70	72	76	85	85	82	85	83	85	89	99	99	98	99	99	98	99
14	JOTHI	72	74	78	78	82	82	84	108	110	112	110	114	118	124	66	66	64	70	68	66	72	80	81	80	83	83	83	89	98	99	99	99	98	99	99
15	SUNDARAM	78	78	84	82	86	88	86	126	134	132	130	134	130	134	72	74	76	76	78	78	76	90	94	95	94	97	95	95	100	100	99	99	100	99	100
16	DINESH	80	86	82	84	86	86	88	132	134	136	132	128	126	128	80	82	80	82	80	84	86	97	99	99	99	96	98	100	99	99	100	100	100	99	100
17	MAHESHWARI	72	78	76	74	76	78	80	128	124	126	128	124	126	122	80	82	86	88	86	84	84	96	96	99	101	99	98	97	100	100	100	99	99	99	100
18	ELUMALAI	82	84	86	84	90	88	86	132	130	128	126	124	122	128	84	82	82	84	80	86	84	100	98	97	98	95	98	99	99	98	99	99	100	100	100
19	SAIRAM	84	78	86	84	88	86	88	110	114	118	124	126	120	128	68	66	64	62	64	66	64	82	82	82	83	85	84	85	100	100	100	100	100	99	99
20	SEKAR	80	84	82	84	88	86	88	126	124	124	126	124	122	126	74	76	78	76	74	78	80	91	92	93	93	91	93	95	98	99	99	98	99	98	99

Patient	GROUP A	HR 3H	HR 4H	HR 5H	HR 6H	HR 8H	HR10 H	HR 12H	SBP 3H	SBP 4H	SBP 5H	SBP 6H	SBP 8H	SBP 10 H	SBP 12H	DBP 3H	DBP 4H	DBP 5H	DBP 6H	DBP 8H	DBP 10	DBP 12	MAP 3H	MAP 4H	MAP 5H	MAP 6H	MAP 8H	MAP 10H	MAP 12H	SPO2 3H	SPO2 4H	SPO2 5H	SPO2 6H	SPO2 8H	SPO2 10 H	SPO2 12H
21	SAMSON	78	74	76	80	78	82	84	118	120	118	124	124	128	126	72	68	74	76	78	76	74	87	85	89	92	93	93	91	100	100	100	99	99	99	98
22	MAHALAKSHMI	82	86	84	86	88	82	88	124	126	124	124	122	124	124	76	74	78	79	80	82	82	92	91	93	94	94	96	96	99	100	99	100	99	99	99
23	RAJESH	74	78	80	82	84	86	86	126	124	122	126	132	130	128	80	78	76	74	76	74	76	95	93	91	91	95	93	93	99	99	98	99	98	99	99
24	KASTHURI	70	76	80	84	84	86	80	102	106	102	104	108	106	108	60	60	62	64	62	64	66	74	75	75	77	77	78	80	99	99	98	99	98	99	98
25	SAKTHIVEL	72	76	78	80	84	86	86	118	122	122	126	124	124	124	70	72	74	76	74	78	76	86	89	90	93	91	93	92	99	98	99	99	99	98	99
26	ANAND	78	76	84	88	86	82	84	116	110	112	110	114	116	118	76	78	84	82	80	82	80	89	89	93	91	91	93	93	99	98	98	98	98	99	98
27	SUKUMAR	80	86	84	86	88	84	82	128	126	124	122	126	130	128	78	78	76	78	78	80	82	95	94	92	93	94	97	97	99	98	99	99	99	98	99
28	GANESH	76	78	82	84	86	90	92	120	122	124	126	122	128	126	76	76	78	78	70	72	72	91	91	93	94	87	91	90	98	98	98	98	98	98	98
29	SAIRABANU	64	66	68	66	68	70	74	114	118	116	116	114	120	124	78	76	72	74	70	72	72	90	90	87	88	85	88	89	99	99	99	99	99	98	98
30	GOPI	74	70	74	76	80	84	86	116	120	122	124	122	120	126	72	74	76	70	72	74	70	87	89	91	88	89	89	89	98	98	98	98	98	98	99
	GROUP B																																			
1	BALAMURUGAN	64	66	70	72	76	74	76	104	106	104	108	110	116	112	62	64	64	64	64	62	68	76	78	77	79	79	80	83	97	98	98	98	98	98	98
2	NIXON	68	64	66	70	72	76	78	106	110	112	110	114	112	114	64	66	66	64	66	68	64	78	81	81	79	82	83	81	98	98	98	98	98	98	99
3	SURESH	70	72	78	76	78	84	86	108	108	106	104	112	118	120	66	70	72	74	72	72	70	80	83	83	84	85	87	87	99	99	98	99	98	99	98
4	MURUGAN	62	62	60	64	68	70	72	120	124	126	126	124	122	124	72	74	76	74	74	76	74	88	91	93	91	91	91	91	98	99	98	98	98	98	98
5	LAKSHMANAN	68	72	74	76	72	72	70	126	124	124	122	120	124	122	68	66	64	68	66	64	68	87	85	84	86	84	84	86	98	98	98	98	98	98	98
6	RAVI	68	70	70	72	74	78	72	118	116	110	120	122	120	122	70	72	74	76	72	72	74	86	87	86	91	89	88	90	99	99	98	98	98	98	98
7	THANUJA	58	64	66	64	64	66	64	130	130	128	126	124	126	126	74	74	68	72	70	70	74	93	93	88	90	88	89	91	98	98	98	99	98	99	98
8	GOWRI	62	64	66	64	64	66	62	128	124	124	126	128	126	130	66	62	64	66	62	68	66	87	83	84	86	84	87	87	99	98	99	100	100	98	99
9	SUKUMAR	60	62	70	72	76	78	78	118	116	114	120	122	120	124	64	64	66	64	66	68	64	82	81	82	83	85	85	84	100	100	99	98	98	98	100
10	SHANKAR	68	66	64	66	68	70	72	110	110	104	106	108	108	114	62	64	66	68	66	64	62	78	79	79	79	80	79	79	98	98	100	100	99	98	98
11	SHANMUGAM	78	76	74	76	74	78	74	102	100	98	100	106	104	108	60	60	64	66	64	68	64	74	73	75	77	78	80	79	98	99	99	98	99	98	98
12	VENKATESAN	80	78	78	82	82	80	82	106	112	116	114	112	108	110	64	62	64	62	66	66	68	78	79	78	79	81	80	82	100	100	100	100	99	98	99

Patient	GROUP A	HR 3H	HR 4H	HR 5H	HR 6H	HR 8H	HR10 H	HR 12H	SBP 3H	SBP 4H	SBP 5H	SBP 6H	SBP 8H	SBP 10 H	SBP 12H	DBP 3H	DBP 4H	DBP 5H	DBP 6H	DBP 8H	DBP 10	DBP 12	MAP 3H	MAP 4H	MAP 5H	MAP 6H	MAP 8H	MAP 10H	MAP 12H	SPO2 3H	SPO2 4H	SPO2 5H	SPO2 6H	SPO2 8H	SPO2 10 H	SPO2 12H
13	SHAKTHI	64	66	68	72	70	72	74	98	102	104	102	104	102	110	60	62	64	68	66	64	68	73	75	77	79	79	77	82	100	99	100	99	98	98	99
14	MALATHI	78	78	80	84	84	76	78	112	114	116	120	122	120	120	68	70	72	72	74	76	78	79	85	86	88	90	91	92	99	98	98	98	98	99	98
15	VELMURUGAN	66	68	68	64	62	66	70	120	120	124	120	120	126	128	78	82	82	84	86	84	82	92	95	96	96	97	98	97	99	99	99	99	99	99	99
16	SURENDRAN	62	60	74	74	74	76	78	108	114	118	120	122	126	124	68	66	64	68	72	74	76	81	82	82	85	89	91	92	98	98	98	98	98	98	98
17	SAKTHI	66	60	62	64	68	70	74	124	124	122	122	124	126	122	72	74	76	78	74	76	74	89	91	91	93	91	93	90	98	98	98	98	98	99	99
18	KALPANA	76	74	80	86	84	84	84	110	120	122	124	126	128	120	72	70	74	78	76	74	72	85	87	90	93	93	96	88	99	99	98	99	98	99	98
19	SEKAR	78	76	77	78	71	70	69	96	100	104	106	110	112	110	62	64	66	68	70	72	72	73	76	79	81	83	85	85	99	98	99	98	99	98	99
20	PRABHU	60	64	66	70	72	74	76	102	102	104	104	106	104	104	58	60	60	62	70	70	72	73	74	75	76	82	81	83	99	99	99	99	99	99	99
21	NATHAN	60	64	64	66	68	66	70	106	106	108	110	106	112	116	74	74	68	70	72	72	72	85	85	81	83	83	85	87	98	98	98	98	98	98	98
22	SHAAHEERA	68	62	64	74	72	74	76	118	110	114	120	120	122	128	76	70	72	74	76	74	74	90	83	86	89	91	90	92	100	100	99	100	100	100	100
23	RAJAN	66	73	76	78	82	84	82	116	118	120	122	124	126	124	68	68	70	72	74	74	70	84	85	87	89	91	91	88	99	98	99	98	99	98	99
24	BABU	74	76	78	74	72	72	70	110	110	118	120	122	120	118	66	62	64	64	66	68	66	81	78	82	83	85	85	83	99	99	99	99	99	99	99
25	MARIMUTHU	70	72	76	80	78	84	84	120	122	124	126	122	124	120	80	82	84	86	84	82	80	93	95	97	99	97	96	93	99	100	99	100	99	99	99
26	SANTHANAM	80	82	84	88	86	90	92	116	118	126	124	128	124	122	70	72	74	76	74	78	76	85	87	91	92	92	93	91	98	99	99	99	98	99	99
27	SHANTHI	70	84	80	82	84	86	89	110	114	112	112	110	112	110	62	62	64	66	68	64	62	78	79	80	81	82	80	78	98	98	99	99	98	98	99
28	SRINIVASAN	76	78	80	82	84	80	86	106	108	108	106	108	110	112	70	72	72	74	72	70	68	82	84	84	85	84	82	83	97	97	98	97	98	98	98
29	SHANKARI	78	78	80	84	88	84	88	118	118	116	112	118	114	120	64	66	64	66	64	68	70	82	83	81	81	82	83	87	98	98	98	98	98	98	98
30	MUTHUKARUPPAN	78	82	84	84	84	90	86	98	108	110	112	106	110	114	58	60	62	68	66	64	68	71	76	78	83	79	79	83	98	97	98	98	98	98	98